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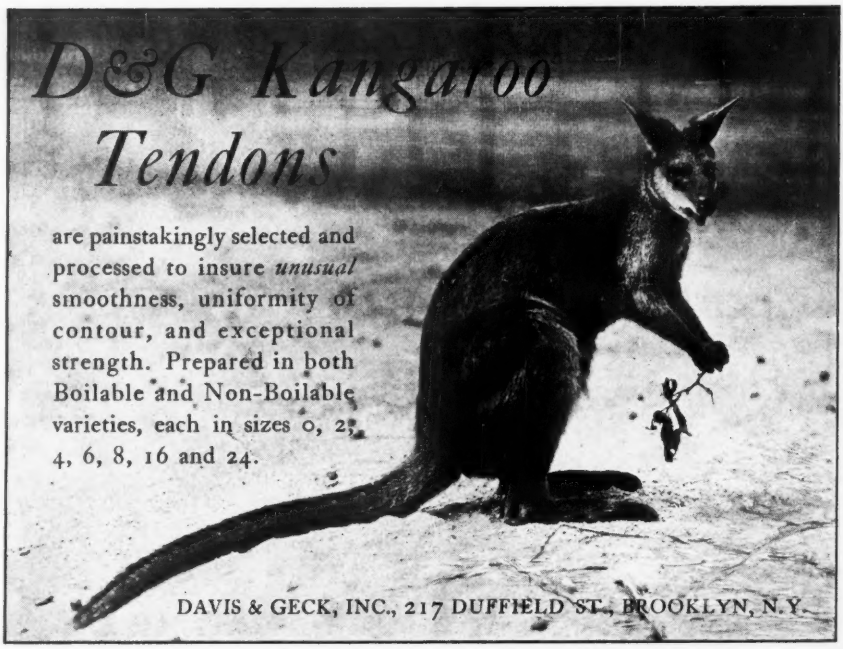
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The Journal of
Thoracic Surgery

VOL. I FEBRUARY, 1932 No. 3

Original Communications

INTRATHORACIC SYMPATHECTOMY FOR THE RELIEF OF REFERRED PAIN*

ETHAN FLAGG BUTLER, M.D.
ELMIRA, N. Y.

PERSISTENTLY recurring pain, localized within the sensory areas of the second dorsal to third lumbar nerves, inclusive, may be caused by sympathetic viscerosensory reflexes. Failing relief from other measures, such pain, if severe enough to justify operative intervention, can be successfully controlled by interrupting the sympathetic afferent fibers. Discussion in this article is limited; first, to referred thoracic pain; second, to the surgery of the sympathetic nervous system in relation to pain only; third, to the operative approach to the thoracic portion of the sympathetic system.

Throughout this discussion the terminology employed by Pottenger[1] will be followed: not only as to vegetative, sympathetic and parasympathetic nervous systems, but also regarding all vegetative or viscerosomatic reflexes, including especially the viscerosensory. In references to operative steps the term ramisectomy will denote section of one or more rami communicantes, white or grey, without, however, either interruption or removal of any part of the sympathetic ganglionated chain. Simple section of the ganglionated chain, without removal of tissue, will be termed sympathotomy. The removal of a portion of the chain with one or more ganglia will be termed sympathetic ganglionectomy, or, for short, sympathectomy. Infiltration of any part of the system by alcohol, procaine, etc., will be referred to as ramus block or sympathetic block, depending on the anatomical structure involved.

According to Behan,[2] the principal causes of thoracic pain are intrathoracic pathology, neuropathology and bone pathology. Even when

*From the Chest Clinic, The Arnot Ogden Memorial Hospital, Elmira, New York. Presidential Address, Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

these conditions can be satisfactorily excluded by modern diagnostic methods, there remains a considerable group of patients who complain of pain in some part of the thoracic wall. These patients are not necessarily disabled. Their condition is not acute. They give a long history of pain at a definitely constant point. Usually there are paroxysmal attacks superimposed on a more or less continuous dull ache. Very often they are diagnosed "pleurodynia" or "intercostal neuralgia," and the possibility of referred pain is ignored. F. T. Lord[3] in discussing the differential diagnosis of pleurisy states, "Although the possibility of pleurodynia or intercostal neuralgia cannot be excluded, this diagnosis amounts to a confession of ignorance concerning the cause of the process." Head,[4] Mackenzie[5] and their followers have offered rational explanations for these pains as sympathetic viscerosensory reflexes.

For a complete discussion of the anatomy and physiology of the vegetative nervous system the reader is referred to Sherrington,[6] Gaskell,[7] Langley,[8] Kuntz,[9] and Pottenger.[1] In this article only a brief explanation of the mechanism of viscerosensory reflexes can be included.

There is ample evidence of the existence of afferent fibers in both sympathetic and parasympathetic fractions of the vegetative nervous system. The afferent neurons associated with the sympathetic system are of spinal origin, and their visceral fibers join the sympathetic trunk through the white rami communicantes between the second dorsal and the third lumbar segments, inclusive. Over these fibers afferent stimuli are constantly passing from the viscera to the central nervous system. Many stimuli, especially those arising in normal viscera, never give rise to conscious sensations. Others, particularly the stimuli arising in diseased viscera, do give rise to conscious sensation, and these sensations are sometimes referred to the viscera in question, but more often referred to some somatic area. Kuntz[9] refers to Mackenzie's work and hypotheses as follows:

"Mackenzie (1910), like Lange (1871-1876), Head (1889), and Faber (1899), regarded the sensory manifestations of visceral disease, which are referred to the somatic segments corresponding to the segments of the spinal cord with which the afferent fibers supplying the viscus in question are connected, as reflex phenomena. He designated them 'viscerosensory reflexes' and explained them mainly on the basis of hyperirritability of the spinal cord resulting from exaggerated visceral stimulation. . . . . He further assumed that when a portion of the spinal cord is excessively stimulated by reason of visceral disease, it may remain hyperirritable for some time, so that the threshold for stimulation for all the nerves arising from this portion of the spinal cord is lowered."

In referring to the work of Szemzo, Kuntz[9] further continues:

"As long as the threshold of stimulation of these cells for peripheral impulses remains sufficiently low, the slightest stimulation of the periph-

eral pain-conducting fibers in the somatic segments in question may elicit painful sensations whose intensity is disproportionate to the intensity of the peripheral stimulus. Under certain conditions, pain may be felt in the corresponding somatic segments even in the absence of appreciable somatic stimulation. On this basis, somatic hyperalgesia associated with visceral disease, in the absence of visceral pain, must be regarded as due to hyperexcitation of the spinal cord cells in question through a flow of visceral impulses whose intensity is below the threshold for visceral pain (Goldscheneider 1923)."

Whatever will increase hyperexcitability of the nervous system will tend to exaggerate these reflexes; hence, they are frequently observed following fatigue, after exposure, in the presence of anemia, and in those physically and nervously below par.

Referred pain mediated in the second dorsal to sixth dorsal segments of the cord will be thoracic. That mediated in the seventh to twelfth segments may be thoracic or located in the anterior abdominal wall. In either case, one or more of the thoracic rami communicantes will form part of the reflex path. If the reflex is to be abolished, either the stimulus must be removed or the path of transmission must be interrupted. Therefore, to quote Scrimger's[10] query, "Given a constant source of irritation in an individual of excessive sympathetic irritability, is it not possible that we should be able to relieve pain by severing the corresponding path of entrance to the cord, in cases when that pain arises from organs showing no gross disease, or where such disease is not locally remediable?"

The foregoing query was prompted by Von Gaza's[11] report of a series of successful dorsal ramisectomies for gastric crises. Barring operations upon the cervical sympathetic ganglia for angina pectoris, these cases are apparently the first instances of operations upon the sympathetic trunk or rami simply and purely for the relief of pain. Since then both Scrimger[10] and Archibald[12] have reported similar successful ramisectomies. Leriche[13] has expressed the opinion: "The surgical treatment of angina pectoris is based upon sensory and not motor considerations." Pieri[14] has relieved pain by ramisection. Flothow[15] has reported an interesting case in which cervical sympathectomy relieved a case of trigeminal neuralgia after operation upon the Gasserian ganglion had failed to produce the desired result. In the case presently to be reported the sole object of operation was to relieve pain, and the selected point of attack was the thoracic portion of the sympathetic trunk and the thoracic rami.

In addition to operations directed at the sympathetic trunk and rami, there are many reports of visceral sympathectomies for relief of pain. In increasing frequency the primary object has been to interrupt the afferent fibers.

Before deciding upon sympathectomy or ramisectomy three rigid tests should be carried out. Von Gaza[11] stipulated two of them; first,

the exclusion of all organic disease which might be amenable either to medical or surgical treatment, and second, diagnostic ramus block with procaine solution. The third test, less important, is the determination of the degree of sympatheticotonia present in the patient. Also, the severity of the pain must be sufficient to justify the operative risk.

CASE REPORT

Mrs. A. R., twenty-nine, white, farmer's wife, on April 22, 1930, gave the following history:

Thirteen years previously, while riding on a wagon, she was seized with a sudden stabbing pain in left chest, so severe that she fainted. The severity rapidly subsided but soreness persisted for three or four days. From that time there was intermittent recurrence of pain in the same area. For the past three months there had been persistent soreness and more severe paroxysmal pain than at any prior time. The paroxysms occurred at intervals varying from five minutes to two hours, but lasted only a few minutes. The pain always occurred on the left side of the chest, starting from "a sore place" high in the left axilla and shooting forward to the anterior midline. It never radiated to the shoulder, arm, neck, back or abdomen. It was aggravated by respiratory effort or lying flat in bed. Slight dyspnea accompanied the paroxysms. There was no history of past or present respiratory, circulatory or digestive disturbances or disease.

In addition to the simple disease of childhood, she had had two attacks of "rheumatism" in the legs, at age twenty-one and age twenty-seven. These attacks lasted only one week and she was not confined to bed. She had had an appendectomy at the age of seventeen; a curettage at twenty-three. Menstrual periods were normal. One child, aged five; no miscarriages. No past history of digestive, urinary, respiratory or circulatory disturbances. Family history negative.

The patient was a well-developed and well-nourished woman. Eye reflexes normal. Many teeth missing and remaining teeth in poor condition. Small firm thyroid but no evidence of hyperthyroidism. No abnormalities could be determined at any point of the vertebral column. Breasts negative. There was superficial hyperalgesia in the left axilla at the level of ribs five, six and seven; also along the course of those ribs. There were tender points at the extreme anterior ends of ribs six and seven. No abnormal findings could be discovered in the ribs or the muscles of the chest wall. Lungs were clear on physical examination. Heart was normal. B.P. 124/86. Abdomen negative. Pelvis negative. Reflexes normal. No edema of extremities.

Fluoroscopic examination of the chest revealed no pathology. No bony, pulmonary, pleural or cardiac pathology was apparent in or suggested by stereoscopic plates of the chest.

The blood Wassermann was negative. Urine examination was negative. Hemoglobin 75 per cent.

Diagnostic ramus block was not carried out. Operation was advised, accepted by the patient and performed at the Arnot-Ogden Hospital on April 30, 1930.

The patient was placed upon her right side in the usual position for major thoracotomy. Nitrous oxide was administered, facilities for positive differential pressure being available. A paravertebral incision was carried downward from the level of rib four to rib six, and thence parallel to that rib, anteriorly and downward to the anterior axillary line. After division of the shoulder girdle muscles, 18 cm. of rib six was subperiosteally resected. The endothoracic fascial

plane was located and the parietal pleura was digitally stripped from the chest wall proper. To gain the necessary exposure, the periosteal bed of rib six was split, the fourth, fifth and seventh ribs were sectioned, posterior to their angles, and a rib spreader was introduced. Two very small pleural tears occurred but caused no embarrassment or difficulty. It was not necessary to section or ligate any intercostal vessels or nerves. As the pleura was stripped posteriorly, the sympathetic trunk and the rami communicantes were beautifully exposed in a bloodless field. Each ramus could be identified easily, so also could the visceral branches, leaving the ganglionated chain to form the greater splanchnic nerve.

No gross lesions were found at operation. There was no bony pathology, there were no tumors, there were no points of pressure along the course of the nerves. The lung could be palpated through the pleural coats and felt normal. There were no pleural adhesions. The structures of the chest wall were in all respects normal.

In the operative procedure no spinal nerve structures were sectioned. The fourth, fifth, sixth and seventh intercostal nerves were subjected to stretching while the rib spreader was in place and were also injected with alcohol at the conclusion of the operation, but the injections were at points distal to, and necessarily remote from, the posterior root ganglia. There was complete excision of the left fourth, fifth, sixth and seventh thoracic sympathetic ganglia with the intervening segments of sympathetic trunk, of the corresponding rami communicantes and of a portion, at least, of the origin of the left greater splanchnic nerve. This involved the resection of afferent fibers from the heart, aorta, lungs and bronchi, pleura, the stomach, liver (possibly), pancreas and small intestine. It involved the resection of efferent preganglionic fibers affecting all the foregoing viscera, as well as the adrenals and the somatic vessels, sweat glands and pilomotor muscles. It involved resection of efferent postganglionic fibers to all the structures under sympathetic control in the corresponding homolateral somatic segments. It involved resection of efferent postganglionic fibers to an unknown extent, but roughly commensurate with the preganglionic fibers. It involved resection of efferent fibers to the middle and inferior cervical ganglia, to the stellate ganglion and to the celiac ganglion.

The immediate operative results would be determined by the sympathectomy, the spinal nerve block and the necessary surgical trauma. There was immediate cessation of the pain for which the patient had sought relief and somatic analgesia in the blocked segments. There was paralysis of the left hemidiaphragm, which began to disappear after a month and a half and was completely gone at the end of eight months. On the day after operation the urine showed 2+ sugar, which had never been present before, and which never appeared in subsequent examinations. There was no "Horner's syndrome." Surgical convalescence was smooth except for a sterile pleural effusion on the operated side which twice required aspiration. There was no respiratory or circulatory embarrassment, nor any gastrointestinal disturbance. She was discharged from the hospital at the end of three weeks quite content, with a well healed wound.

The patient was observed at frequent intervals, and at the end of a year she willingly returned for a complete study. Late results could be determined only by the sympathectomy. There had been no recurrence of the pain for which she had sought relief. She was comfortable and in no wise incapacitated. Blood pressure, urine, blood counts, gastric acidity were all normal. Blood Wassermann continued negative. Blood sugar was low, 77.2 mg. per 100 c.c.

The electrocardiogram was reported "Left preponderance. Third lead shows incomplete block (bundle of His)." There was no sweating in the left axilla or over the left chest wall in the sympathectomized zone. The most interesting re-

sults were obtained by bronchoscopy and by gastrointestinal x-ray series. There was edema of the tracheal and bronchial mucosa and collapse of those tubes, such as is seen in asthmatics, although there were no such symptoms. Adrenalin did not affect this condition, neither did atropine. The esophagus was normal in its appearance and behavior. There was neither cardiospasm nor pylorospasm. The stomach emptied rapidly. The only outstanding evidence of loss of sympathetic control was in the absence of sweating in the left chest wall, the bronchoscopic findings and the rapid emptying of the stomach. None of these were attended by subjective symptoms on the part of the patient.

On the basis of the foregoing reports and studies, it is reasonable to say that the thoracic sympathetic trunk and its branches can be interrupted without undue risk to the patient. This is not the place to speculate as to possible indications for such operative intervention, but it is appropriate to discuss the avenues of approach that are open to the surgeon.

Except for removal of intrathoracic sympathetic ganglioneuromata and the removal of the stellate ganglion by cervical approach in connection with cervical sympathectomy for angina pectoris, there, apparently, have been no reported operations upon the thoracic sympathetic trunk or its branches prior to Von Gaza's ramisectomies. Von Gaza[11] employed a direct, paravertebral approach; erector spinae muscle bundles being retracted medially, the intercostal nerves directly located in their interspaces, followed back until the rami communicantes were visualized, and then, the end objects having been obtained, the rami were sectioned and the operation concluded. Such an approach was essentially extrathoracic, and could not afford a good view of the sympathetic trunk. Such an approach would permit of bilateral ramisectomy should it be desired and would carry no risk from intrathoracic manipulation. Scrimger and Archibald employed the same operative approach and evidently found it adequate. In 1924 Henry[16] suggested superior, posterior, extrapleural mediastinotomy through the periosteal bed of the second rib as the optimum approach to the stellate ganglion. At this level the shoulder girdle muscles are heavy and the operative field within the thorax is very limited. Coincident bilateral attack through one midline incision is possible. This approach did not prove to be the best for stellate ganglion, and certainly would not be of any value for any segment below the third. In 1927 Pieri[17] proposed a limited extrapleural posterior mediastinotomy as a rational approach to the thoracic sympathetic trunk at any desired level. Pieri advocated paravertebral resection of short segments of three ribs at the desired level, complete section of the intervening intercostal muscle bundles, together with the accompanying artery and vein, but not section of the nerve. The endothoracic fascial plane was located and the pleura stripped from the chest wall to expose the rami and the ganglionated trunk. Such an approach would afford a fair, though limited, view of the sympathetic chain. The operative field would probably be dry. The rapidity of approach would depend

largely upon the surgeon's familiarity with the anatomical area and structures involved. The risk to the patient would vary inversely with the surgeon's familiarity with the special requirements of thoracic surgery. Coincident bilateral operation at the same or different levels would be a formidable, but not necessarily impossible, procedure. In 1928 Rossi[18] suggested and described an inferior, posterior, infrapleural mediastinotomy through the twelfth rib, particularly as an approach to the splanchnic nerve. It would afford a very limited operative field, but could permit coincident bilateral attack, granted its permissibility. The risk from open operative pneumothorax and other purely thoracic complications would be minimal. Usefulness would be absolutely limited to the lowermost thoracic segments and the splanchnic nerves. In July, 1928, Adson[19] utilized superior, posterior, extrapleural mediastinotomy, through the second rib in approaching the stellate ganglion and proved that coincident bilateral operation was feasible and safe for the patient. The optimum level was changed, however, to the first rib in 1930 and it may fairly be said that Adson[20] has arrived at a satisfactory surgical approach to the stellate ganglion. So far as a search of the literature has revealed, there are no other reported or suggested operative procedures.

Intrathoracic vagus fibers have also been subject to operative attack. Phillips and Scott[21] have reported one such case, operated upon in 1928, the desired portion of the vagus being exposed by a wide, transpleural, right, posterior thoracotomy.

Necessarily the sympathetic trunk must be approached posteriorly, and necessarily some variety of posterior mediastinotomy must be employed. In all the previously reported operative approaches a limited portion only of the sympathetic system could be exposed. By wide extrapleural, exploratory thoracotomy, as described in this article, the entire thoracic chain on one side of the body can be rapidly and completely exposed. There is a minimal amount of trauma to the structures of the chest wall proper. There is a minimal amount of bleeding and consequent obscuring of structures in the depths of the wound. It is necessarily a unilateral procedure, and the surgeon should be well versed in the special principles of thoracic surgery. So long as the procedure is extrapleural, there is little risk from variations in intrathoracic pressure. There should be available facilities for applying differential pressure in the event of a pleural tear. Special skill should be expected of the anesthetist. The level of the approach can be varied to fit individual requirements, but an approximately midthoracic approach will allow rapid, accurate, practically bloodless exposure of the entire chain.

CONCLUSIONS

1. Thoracic pain, in the absence of definite intrathoracic pathology, neuropathology or bone pathology, may be due to a sympathetic vis-

cerosensory reflex. If so, interruption of the reflex arc will relieve the pain.

2. Complete unilateral removal of the fourth, fifth, sixth and seventh dorsal ganglia, together with section of the corresponding rami and the origin of the greater splanchnic nerve, is not incompatible with life, nor did it cause disability in the reported case.

3. Posterior, extrapleural exploratory thoracotomy affords the optimum surgical approach to the thoracic portion of the sympathetic chain, provided only that it be carried out in accordance with the recognized principles of thoracic surgery.

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RECENT ADVANCES IN OUR KNOWLEDGE OF THE AUTONOMIC NERVOUS SYSTEM*

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ST. LOUIS

TO THE thoracic surgeon the autonomic nervous system occupies a position of considerable interest. Some of his successes and not a few of his unsolved problems bear a close relationship to it. It seems pertinent, therefore, that from time to time one should review functional and morphologic developments in our knowledge of this system, in order that we may more rationally evaluate advocated procedures and possibly see our way to the establishment of new ones.

The function of the autonomic nervous system has generally been regarded as a regulatory one. The recent work of Cannon (1930) and his associates has emphasized this. They have shown that the autonomic nervous system is concerned with the maintenance of a constant internal environment for the bodily organs, compensating external influences (homeostasis). It accomplishes this by acting on the heart, smooth muscles, and glands. Cannon has consequently termed it the interofective system in contrast with the exterofective or voluntary nervous system. He has shown that animals may live without a sympathetic nervous system provided that they are maintained in the sheltered environment of the laboratory. This doubtless will afford surgeons a feeling of security in attacking the sympathetic nervous system by surgical procedures. It should not be forgotten that the patient in most instances cannot be so completely shielded from external influences as were Cannon's experimental animals. Consequently he may suffer a handicap in his environmental adjustments. Not without clinical significance also is the work which indicates that adrenalin has an action similar to the sympathetic nerves and may function directly on the tissues without the intervention of the extrinsic nerves themselves.

Following the announcements of De Boer (1913), Hunter (1925) and others with regard to the effect of the sympathetic and sympathectomy on striated muscle, surgeons have performed operations for the relief of spastic states. The results reported are voluminous and have varied greatly in nature, depending mostly upon the optimism of the operator. Recent experimental work by various investigators including Tower (1926), Hinsey (1927), Cannon (loc. cit.), and others

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may now be said to have conclusively demonstrated that sympathectomy has no direct effect of appreciable significance on the histology or function of an ordinary muscle fiber. It seems, therefore, permissible to conclude that sympathectomy is not a measure to be utilized in operations for the relief of pathologic states resident within skeletal muscle. The domain of the surgeon of the sympathetic nervous system remains confined to the heart, smooth muscle, and glands.

Of more immediate interest to the thoracic surgeon is the progress which has been made in the treatment of Raynaud's disease. Here again the worker in the fundamental sciences has been largely responsible for the successes of the surgeon. Kuntz (1927) demonstrated that the extirpation of the inferior cervical and first thoracic ganglia or section of the communicating rami joining the brachial plexus did not result in complete sympathetic denervation of the vessels of the upper extremity in a large percentage of cases because in such cases sympathetic fibers join the brachial plexus from the second thoracic sympathetic ganglion via the inconstant intrathoracic ramus running from the second to the first thoracic nerve. Whenever this ramus joins the first thoracic nerve proximal to the origin of the first intercostal ramus it constitutes a pathway through which sympathetic fibers derived from the second thoracic ganglion may join the brachial plexus. Adson and Brown (1928) and others have found that extirpation of the inferior cervical sympathetic ganglion, together with the first two thoracic ganglia, will result in complete success in the treatment of Raynaud's disease whereas extirpation of the inferior cervical sympathetic ganglion and cervical ramisection alone does not always do so.

A reasonable hypothesis concerning the mechanism of relief by surgical intervention in Raynaud's disease might be stated thus: The removal of the sympathetic components eliminates the efferent fibers whose stimulation results in spastic crises of the blood vessels. These crises may be central or peripheral in origin. Pain is relieved because ischemia no longer develops to result in stimulation of sensory nerve endings. Some visceral afferent paths are eliminated when the sympathetic fibers are cut.

One of the objects of this presentation is to familiarize you with a new method of study of the autonomic nervous system which is being employed at Washington University. (Heinbecker, 1929; Bishop and Heinbecker, 1930.) Its distinctive feature concerns the use of the cathode ray oscillograph in association with an amplifier as a recording device. The technic is now an established one but for those whose interests make them more familiar with clinical, rather than with the physiologic literature, a brief explanation will be given. The cathode ray oscillograph is generally employed for electrical measurements, but it has been only recently applied to physiologic studies (Gasser, Erlanger and Bishop). It is a thermionic vacuum tube of a type in which

a beam of electrons is projected onto a fluorescent screen painted on the end of the tube. The electron beam on its course to the screen passes between two pairs of plates set at right angles to each other. A potential applied to the vertical pair of plates deflects the beam in the horizontal plane and the focused electrons move across the screen as a line. The speed of this movement across the screen is controllable and measurable. The line serves similarly to a base line in a kymographic record. By means of a circuit breaker or double key a stimulus may be synchronized with any point in this deflection. This stimulus, together with the resultant action potential from the nerve, is applied to the other pair of plates which cause a deflection of the electrons beam as right angles to the base line.

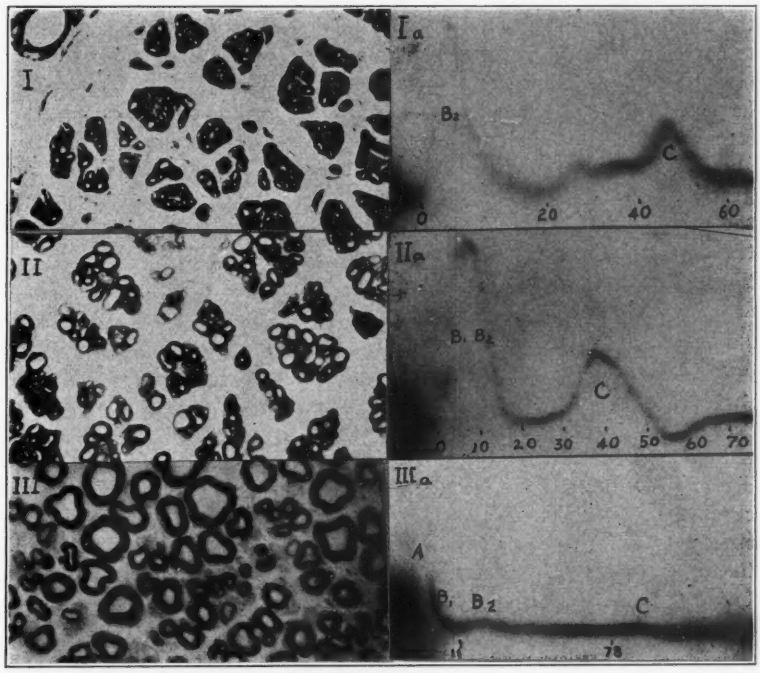

Fig. 1.—Shows the method of correlating fiber type with action potential form in nerve. All sections stained with osmic acid, magnification 880X. Note in cross-section I two types of fibers unmyelinated and small myelinated. Note two waves marked B_2 and C in the action potential Iâ derived from this nerve. In cross-section II there are three fiber types. In addition to the unmyelinated and small thinly myelinated there are larger myelinated fibers afferent in type (vagus nerve). Note the corresponding action potential IIâ shows three waves marked B_1, B_2 and C. Cross-section III from the sciatic shows four fiber types, large thickly myelinated somatic motor and sensory, smaller relatively thinly myelinated, small thinly myelinated and unmyelinated fibers. The action potential IIIâ shows four waves marked A, B_1, B_2 and C.

Some nerve trunks are composed almost entirely of unmyelinated fibers and from these we learn that such fibers usually travel less than a meter per second. This permits us to know that the last wave in action potential, where there are unmyelinated and small thinly myelinated fibers, is derived from the unmyelinated fibers. The first wave in the potential from a nerve as shown in Iâ has its first wave traveling too rapidly to be produced by unmyelinated fibers. The first wave is essentially from the myelinated fibers.

It is known to you that when a nerve fiber becomes active there is a potential change at the site of activity. This activity is propagated along the nerve fiber. Recording electrodes placed on the nerve will serve to convey the potential change of activity to the amplifier-oscillograph combination and cause a deflection of the electron beam. Inasmuch as different fibers within a complex nerve trunk have different conduction rates it is readily understood that when a group of fibers are activated by an electrical stimulus and the activity propagated along the axons, those in which the propagation is most rapid will produce a change in the recording electrodes before the more slowly conducting axons. As a consequence from a complex nerve trunk the complete conducted action potential will consist of a series of waves. By correlation between the recorded potential and the cross section of the nerve trunk as revealed in osmic acid sections it has been possible to correlate with each of the various waves a specific fiber type which is essentially responsible for its production, (Fig. 1). The fiber types have also different thresholds so that by gradually increasing the strength of stimulating current one wave after another may be elicited and by observing the effect of each wave as it appears on the oscillograph in the intact animal it is possible to determine the function of the various fiber types. Besides responses to electrical stimuli, spontaneous responses can also be recorded.

COMPOSITION OF AUTONOMIC NERVE TRUNKS AND THE PROPERTIES OF THEIR AXON TYPES

Autonomic nerve trunks histologically studied (osmic acid) reveal three fiber types, a larger and relatively thickly myelinated one, a small thinly myelinated one and an unmyelinated one. Most nerve trunks contain all three types but some contain only two. By a number of procedures it has been shown that the first type is afferent in character (Heinbecker, 1930). The properties of its potential are strikingly different from the properties of the potentials derived from the other two types. The second and third types so far have been found to be entirely efferent in function, and autonomic in distribution in general. The potential derived from the afferent fiber type is similar in its properties to the potentials derived from ordinary somatic nerve fibers. It has, therefore, a rapid conduction rate, a short absolutely refractory period, a short chronaxie and a short duration of potential. The small thinly myelinated and unmyelinated fibers have in contrast properties which permit them to be readily distinguished from those of the first type. They have a slow conduction rate, a long absolutely refractory period, a long chronaxie and a potential which rises slowly to a maximum. The properties of these fibers are 5 to 10 times slower than the properties of the somatic nerve fibers. By way of illustration one can state that in man the first type travel about 15

meters per second, the second type 6 to 8 meters per second, and the third type half a meter per second or less. The significance of these marked differences in properties may not at once be apparent to you. Aside from the fact that we have here evidence for the first time of an absolute difference in axon type of somatic and autonomic nerve fibers (Heinbecker, 1929) we have also the means of recognizing these two fiber types anywhere in the body, tracing their course or determining their function. We are able to do this because it has been possible to correlate the form of a conducted potential derived from an autonomic nerve fiber with its histologic content. By this means it has been possible to show that the force of the heart beat is lessened through the activity of the small thinly myelinated fibers in the vagus nerve and its rate is primarily altered by the activity of the unmyelinated fibers (Heinbecker, 1930). In the sympathetic nerves to the heart it has been shown that both the force of the beat and its rate are increased by unmyelinated fibers. No differences have been found in the properties of sympathetic and parasympathetic fibers of the same anatomical type. Likewise no differences in properties have been found in similar fiber types preganglionically and postganglionically, the response depending only on the end-organ.

STUDIES OF THE SUPERIOR CERVICAL GANGLION

The fibers pre- and postganglionically have been studied as to type, potential properties, and function, (Heinbecker and Bishop, 1931). Studies of the fiber types in various animals have revealed that preganglionically there are always two types of autonomic efferent fibers, one myelinated, the other unmyelinated. No efferent fibers have been found in mammals to pass through this ganglion without synapsing. Postganglionically we have found in certain animals (cat and monkey) both myelinated and unmyelinated efferent fibers. In the rabbit only unmyelinated efferent fibers have been found postganglionically. It is apparent, therefore, that we are unable to support the hypothesis of Gaskell in which he states that all postganglionic sympathetic fibers are unmyelinated, (Fig. 2). While the situation in man has not been investigated functionally, all our morphologic and functional studies on human nerves have indicated a striking similarity with those of the monkey. There is little doubt, therefore, that Gaskell's hypothesis does not hold in man.

Ranson and Billingsley (1918) suggested that one of the chief functions of the outlying neurones of the sympathetic nervous system is to multiply the discharging paths. Querido (1924) at Harvard has stated that these neurones are capable of discharging at an optimum frequency as a result of preganglionic stimulation of different frequency. Our own observation made directly on the ganglion by a method permitting the actual observation on the oscillograph of both pregan-

glionic and postganglionic responses fails to indicate any evidence for such an independent rhythm on the part of the ganglion cells. Our observations have been in agreement with the general impression that no reflexes take place in the outlying sympathetic neurones, but that a given preganglionic fiber may discharge an impulse to several postganglionic fibers.

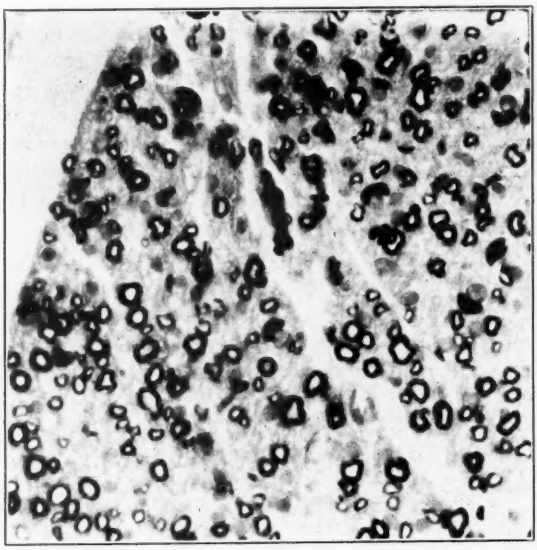

Fig. 2.—Osmic acid cross-section cat postganglionic cervical sympathetic trunk. In this nerve there are three fiber types, unmyelinated, small thinly myelinated and medium size more thickly myelinated. The latter are afferent in type, the first two are typically efferent. This figure shows that Gaskell's hypothesis does not hold in the cat. Functional experiments have shown that all the efferent fibers synapses in the cat superior cervical ganglion so that the small thinly myelinated fibers in this nerve section are efferents.

A STUDY OF THE AFFERENT AND EFFERENT FIBER PATHS TO THE HEART AND AORTA

The above outlined study on nerve trunk composition and sympathetic ganglia has served as a basis for study of the fiber paths and fiber types concerned in the problem of angina pectoris. It has been found that afferent fibers are present in all three sympathetic cardiac nerves. The afferent fibers traveling in the *superior cardiac nerve* (Fig. 3) pass through the superior cervical ganglion to end in the ganglia of the vagus nerve. The presence of these fibers would explain in part at least the effects obtained surgically by removal of the sympathetic ganglion. In animals (turtle, cat, and rabbit) it has been found that there *may be* a separate depressor nerve associated either with the vagus or with the sympathetic trunk. When such a nerve is associated with the sympathetic trunk its fibers have been invariably

found to pass directly through the superior cervical ganglion without synapse to end in the vagus ganglia. It is always complex in structure, afferent fibers being mixed with the types typically efferent elsewhere. Studies of the sympathetic and the vagus trunks in four human beings both functionally and histologically have failed to reveal the presence of any organized group of fibers which could be described as a

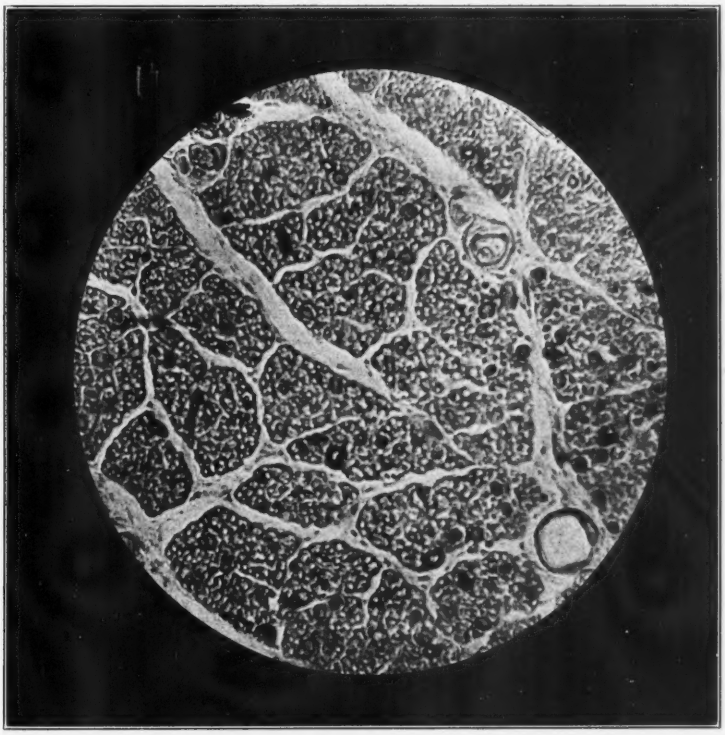

Fig. 3.—Osmic acid cross-section monkey superior cardiac nerve. Nearly all fibers are unmyelinated efferents except a few myelinated fibers of the afferent type. These afferent fibers have properties of the quick type like somatic fibers.

depressor nerve trunk. This does not in any way indicate that such fibers are not present but that either they mix with other fibers in the general vagus nerve trunk, or that the depressor nerve is not significantly different in fiber composition than other divisions of the vagus. Consequently it does not seem possible for surgeons to remove a depressor nerve in human cases of angina pectoris. Our observations are in agreement with those of other investigators who find afferent fibers in the middle and inferior cardiac nerves. Nerves to the heart connecting with the first six or seven thoracic ganglia have been found to

contain fibers of the afferent type. The vagus nerve contains many afferent fibers. It is apparent that to eliminate afferent paths from the heart and aorta both sympathetic and vagus pathways would have to be eradicated (Fig. 4).

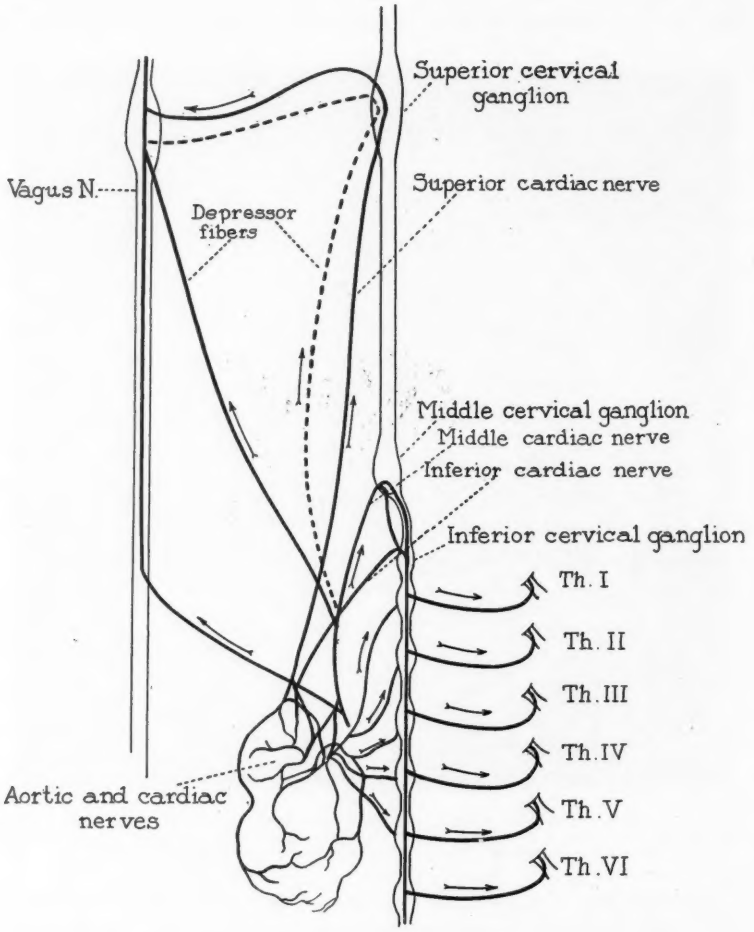

Fig. 4.—Figure to show the course of afferent fibers found in nerves connected with the heart and first portion of the aorta. Note that afferent fibers are present in the superior cardiac nerve and these pass through the superior cervical ganglion to the vagus ganglion. This explains why, in cases of angina pectoris, removal of the superior cervical ganglion alone can have an effect. The course of the depressor fibers is generally along with those of the vagus nerve. In some animals they form a separate trunk. In man in the cases studied (4) they have been combined with the vagus trunk. In some animals (cat and turtle) the depressor nerve may also in some instances be associated with the cervical sympathetic trunk (dotted line in figure). Then its fibers simply pass through the superior cervical ganglion to the vagus ganglion. This arrangement has so far not been found in man. There are afferent fibers in the vagus trunk and also afferent fibers in nerves coming from the first 6 or 7 thoracic sympathetic ganglia. These fibers pass to the corresponding thoracic spinal ganglia.

In all autonomic nerve trunks to the heart and aorta fibers which are typically efferent in type have been found histologically and in many cases demonstrated functionally. In this connection reference is to be made to the experimental work of Porker (1896), Maass (1899), Wiggers (1909), Greene (1931) and others in which they have shown that fibers are present in the vagus nerve of animals whose *function is to cause constriction of the coronary vessels.* Likewise Maass, Greene and others have demonstrated that sympathetic nerve trunks to the heart contain fibers which cause a dilatation of coronary vessels. Nerve fibers have been shown to exist in the coronary vessels of man, but the functional nature of these fibers must be assumed from animal experimentation. Our own investigations in the cat and rabbit have demonstrated that postganglionically it is the unmyelinated fibers in the cervical sympathetic nerve trunks which are responsible for skin vascular constriction.

Even though complete unanimity of opinion concerning the pathogenesis of angina pectoris cannot be assumed, evidence for a vascular spasm of the coronaries or vasa vasorum of the first portion of aorta being part of the mechanism of the disease is now generally accepted. It seems not inconsistent to conclude, therefore, that the procedure of choice for *curative* purposes would be one which eliminated the vagus constrictor fibers. The sympathetic efferents beneficially would be left alone. The surgery of angina pectoris so far has largely been directed against the visceral afferent fibers of the sympathetic because they happen to be pain fibers. These painful influences may in many instances simply be an expression of the abnormal activity of the vasoconstrictor fibers (resultant ischemia). In other instances, especially where marked pathology exists, the afferent impulses may be responsible for initiating the reflex constriction. More and more evidence is accumulating that in man functional disturbances of organs may result from central influences, themselves directly or indirectly initiated, and acting through the hypothalamus. Whenever possible our efforts in surgery should be aimed to be curative rather than to give mere symptomatic relief. We cannot afford to forget that the heart, lungs and other organs have a dual innervation. To attack always the sympathetic system because it is available without regard to established knowledge of physiology does not seem commendable. It is granted that measures giving symptomatic relief must be employed at times, but we should not rest with these.

Some apology is made for a lack of completeness in our summary of advances in our knowledge of the form and function of the autonomic nervous system. Enough has been said to indicate our faith in the principle that it is always advantageous to apply our knowledge of physiology to the problems of surgery. When such knowledge is lacking, it is better to seek it first rather than to operate to learn the effect of a procedure on so complex a mechanism as the human body.

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FACTORS ALTERNATING INTRAPLEURAL PRESSURE AND THEIR CLINICAL SIGNIFICANCE*

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THE fact that there is a "negative" (subatmospheric) pressure in the potential pleural space is well understood. Also it is generally accepted that in the normal living animal the visceral pleura is in accurate and intimate apposition everywhere to the parietal pleura except that there is a small amount of fluid which acts as a lubricant between the two surfaces. The negative pressure is present not only in the intrapleural space but also in the mediastinum, and therefore affects all the structures within the thorax. Some investigators[1, 2] have claimed that because the pleural space is only potential, a negative pressure cannot exist. However, this question is of academic interest only, since they admit that there is a negative tension within the chest on its contents and that as soon as a trocar is inserted into the pleural space a negative pressure can be demonstrated because a small amount of air necessarily is sucked in.

The importance of this negative pressure on the circulation and its relation to the elasticity of the lungs was appreciated by Carson[3] as long ago as 1820. This ingenious investigator inserted a cannula into the trachea of different animals and on a water manometer measured the increase in pressure upon opening the thorax and collapsing the lungs. However, Donders[4] in 1850 was apparently the first to demonstrate that respiration produces a change in intrapleural pressure and that it is more negative at the end of inspiration than at the end of expiration. Using a method similar to Carson's, he introduced a cannula into the trachea of a cadaver and after opening the thorax measured the resultant pressure. This he assumed to be the pressure at expiration. By artificially distending the lungs with air he found that at inspiration the pressure was higher and therefore he assumed that the intrapleural pressure would be more negative. In 1877 d'Arsonval[5] attempted to measure intrapleural pressure directly by inserting a trocar into the pleural cavity of a dog. Luciani and Rosenthal[6] (1880) demonstrated by measuring the intraesophageal pressure in various animals that the negative pressure within the thorax is re-

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flected in the esophagus, and later Rosenthal and Leube[7] showed this to be true in a human being. For a more complete review of the early work in this subject the report of Heynsius[8] should be consulted.

Meltzer[9] (1892) attempted to ascertain the degree of distention in different parts of the lung by measuring the intrathoracic pressure in different parts of the mediastinum. His method was to introduce a catheter through the pharynx into the posterior mediastinum. This catheter was connected to a tambour and water manometer. With this method he noted marked differences in pressure in different parts of the mediastinum.

According to Emerson[10] the first successful determination of intrapleural tension in normal man was made by Aron.[11] In thirty-six estimations on one subject he found the average tension at the height of quiet inspiration to be –4.6 mm. mercury, and at the height of expiration to be – 3.02 mm. mercury. Aron also noted that when a patient was sitting up in bed or on a chair the pressures were more negative than when lying down. He demonstrated that in open and closed pneumothorax in an experimental animal (rabbit) the effects of the pneumothorax can be demonstrated on the opposite side.

Nearly all of the recent experimental work on intrapleural pressure has had to do with the relation of intrapleural pressure to pneumothorax. For a review of this phase of the work the reader is referred to the articles by Graham,[12] Snyder,[13] and Duval.[14] In general it has been shown that the intrapleural pressure is affected by disease within the lung, especially atelectasis,[15] disease within the pleural cavity and elsewhere in the chest, and by changes of position. A very complete study has been made of the effects of pneumothorax.[12] It has also been suggested that the pressure of the two sides of the chest is not necessarily the same and that it varies in different parts of the same side of the chest. There has been a good deal of discussion of the effect and importance of intrapleural pressure on the cardiovascular system. As far as we can find there has been no systematic study of various other factors that may affect intrapleural pressure or the relationship between intrapleural pressure and thoracic girth. This report concerns a few of these factors.

METHOD

The method we used in this study is illustrated in Fig. 1. Dogs were used in all experiments. Some of them were trained so that no anesthesia was necessary, but in many instances sodium amytal* was used intraperitoneally as an anesthetic agent. The trocars used were number seventeen gauge needles, and the tubing from these trocars was

*We wish to acknowledge the assistance of the Eli Lilly Company in donating the ampules of sodium amytal used in these experiments.

branched so that both manometric and kymographic records could be obtained from each side of the chest, as is shown in Fig. 1. In all the early experiments pressure readings were taken from both sides of the chest. It was found in all instances, even when partial pneumothorax was produced, that the effect on the intrapleural pressure on

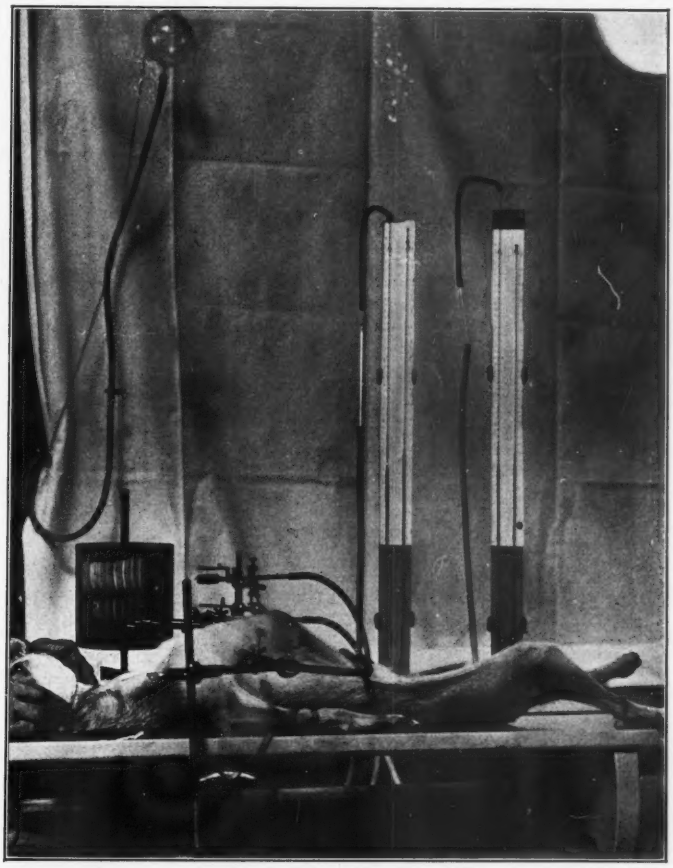

Fig. 1.—Method used to study factors altering intrapleural pressure. Dog not under anesthetic. Tracing on kymograph shows increased (more positive) intrapleural pressure following administration of epinephrin.

both sides was qualitatively similar. Therefore in the later experiments we frequently recorded intrapleural pressure on only one side of the chest to save space on the drum and apparatus. The apparatus was made as air-tight as possible. Occasionally the carotid blood pressure was recorded also. In a number of the experiments a pneumograph (Dr. Ellis') was used to record the expansion and contraction

of the chest. Frequently this was checked by actual measurements with a steel tape. When the effect of gases on intrapleural pressure was studied, the gas was administered in many cases through a cannula fitted tightly into the trachea through a tracheotomy opening.

We realize that there are many objections to our method of recording intrapleural pressure, and it probably does not give us the true

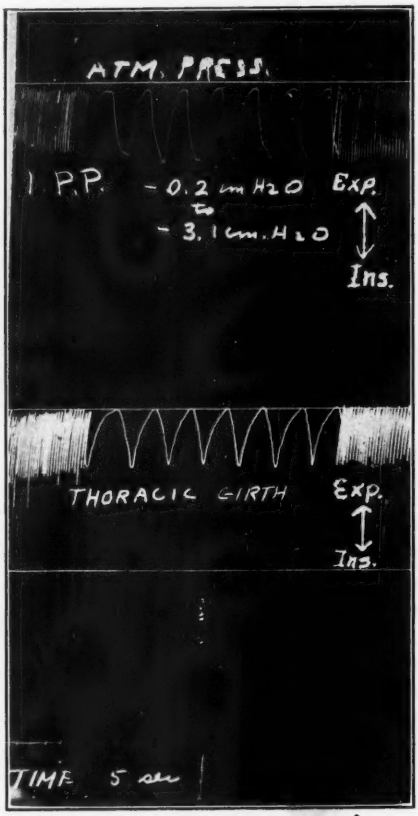

Fig. 2.—Kymographic tracing of intrapleural pressure and respiratory movement in a normal unanesthetized dog. The down stroke in both tracings is made on inspiration and the up stroke on expiration. Note that the intrapleural pressure (I. P. P.) is subatmospheric.

pressure in the pleural cavity. We found that we could not check intrapleural pressure from day to day in the same animal or sometimes even during any one day. We confirmed that the pressures in different parts of the chest vary. However, we believe that with the trocar in one place during any one experiment, the relative results can be considered trustworthy.

An example of what may be considered a normal curve is shown in Fig. 2, which is a tracing of the intrapleural pressure and of the expansion and contraction of the chest, which is designated "thoracic girth." This tracing was made on a normal, unanesthetized dog.

Atmospheric pressure is obtained by opening the rubber tube connected with the tambour as the drum revolves. This can be done at the beginning or at the end of the experiment. The horizontal line of the pneumograph is obtained in the same way and serves merely as a control line. In both tracings, that of the intrapleural pressure and that of the thoracic girth, the down stroke of the graph is obtained with inspiration and the up stroke with expiration. It will be noted that the intrapleural pressure is entirely subatmospheric both at inspiration and at expiration, and maintains a fairly constant level, and that the tracing of the expansion and contraction of the chest also remains fairly constant when the animal is quiet. The length of the stroke means little in comparing the different graphs since it depends on the tightness of the rubber diaphragm of the tambour which may vary in different experiments. The fulcrum of the tracing needle was the same in all instances.

TABLE I

Dog—Wt. 6 kilos. No anesthesia. Intrapleural pressure readings after injection of 2 c.c. epinephrin hydrochloride 1/1000 subcutaneously.

	RIGHT CHEST		LEFT CHEST	
	I.P.P. in cm. of H_2O	Mean I.P.P. in cm. of H_2O	I.P.P. in cm. of H_2O	Mean I.P.P. in cm. of H_2O
Normal average	+0.6 to −8.4	−3.9	+2.0 to −8.6	−3.3
5 min. after epinephrin	+2.6 to −8.4	−2.9	+3.4 to −8.6	−2.6
8 min. after epinephrin	+2.6 to −8.8	−3.1	+3.4 to −8.2	−2.4
9 min. after epinephrin	+3.6 to −8.4	−2.4	+3.2 to −8.2	−2.5
15 min. after epinephrin	+3.6 to −9.0	−2.7	+4.2 to −8.2	−2.0
17 min. after epinephrin	+3.6 to −8.4	−2.4	+3.8 to −8.2	−2.2
24 min. after epinephrin	+4.4 to −8.8	−2.2	+5.8	
29 min. after epinephrin	+4.0 to −8.8	−2.4		
1 hr. after epinephrin	+5.6 to −7.8	−1.1		

TABLE II

Dog—Wt. 9 kilos. Anesthesia sodium neonal, 450 mg. intraperitoneally. Intrapleural pressure readings after intravenous injection of 0.4 c.c. epinephrin hydrochloride 1/1000.

	I.P.P. Right Chest in cm. H_2O	Mean I.P.P. in cm. H_2O
Normal	−3.6 to −7.0	−5.3
3 min. after epinephrin	−3.6 to −7.2	−5.4
6 min. after epinephrin	−3.4 to −7.2	−5.3
8 min. after epinephrin	−2.6 to −6.6	−4.6
10 min. after epinephrin	−1.6 to −5.6	−3.6
11 min. after epinephrin	+0.2 to −5.6	−2.7
13 min. after epinephrin	+3.0 to −5.6	−1.3
15 min. after epinephrin	+5.4 to −5.6	−0.1

EFFECTS OF DRUGS

Bronchodilating Drugs.[16]—Epinephrin and atropine both cause bronchodilatation; the former is a typical stimulant of the thoracolumbar autonomics, and the latter acts by peripheral paralysis of the craniosacral autonomics. Both of these drugs are used commonly in clinical medicine and their other effects are well known. Following the administration of these drugs in the proper dosages, we found uniformly that the intrapleural pressure became more positive, both at inspiration and expiration. This is well illustrated in Fig. 3 and Tables I, II, and III. Further, it was found that as the intrapleural

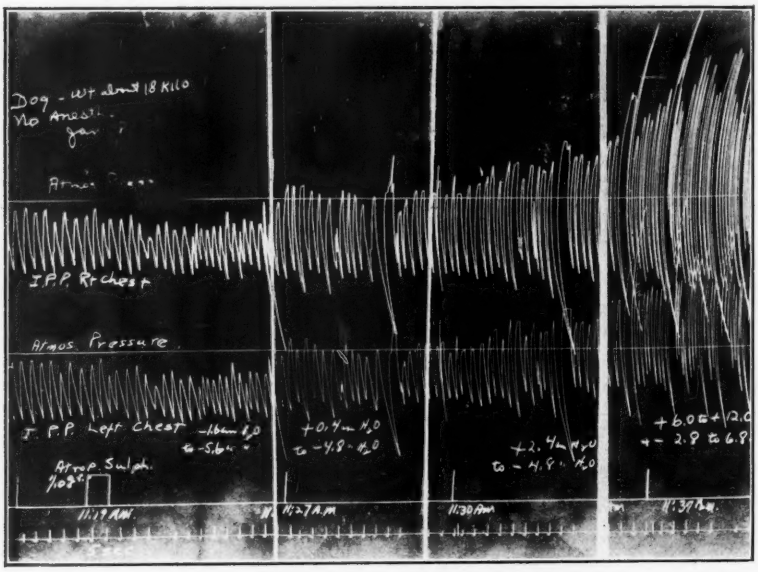

Fig. 3.—Kymographic record of intrapleural pressures of both chests of unanesthetized dog following subcutaneous administration of six milligrams of atropine. Horizontal lines are the atmospheric pressures in each chest. Above the horizontal lines the pressures are greater than atmospheric, and below they are subatmospheric. The figures on the graph indicate actual pressure measurements in centimeters of water.

pressure became more positive the mean thoracic girth decreased (Table IV).

If we assume that the animal used in these experiments is in the resting position and that the respiratory exchange is more or less constant, these findings might be expected, namely, that with bronchodilatation the intrapleural pressure becomes less negative and the mean thoracic girth is decreased. Bronchodilatation results in lowered resistance to movement of air into and out of the lungs, and less than normal expansion of the chest is required for the inspiration of the same amount of air. Therefore the intrapleural pressure should

be less negative and the mean thoracic girth should be decreased. In these instances the breathing is taking place with the chest relatively in the expiratory position. However, we found that there may be

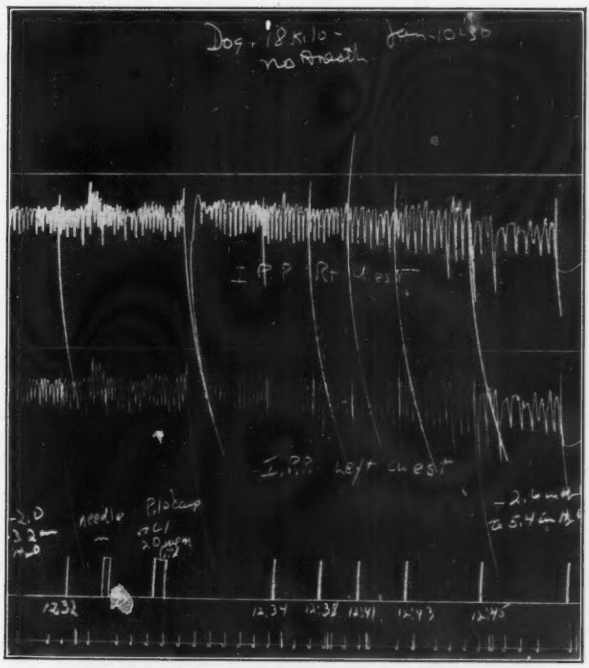

Fig. 4.—Kymographic record of intrapleural pressures of both chests of an unanesthetized dog showing effects of pilocarpine. For explanation see Fig. 3.

TABLE III

Dog—Wt. 18 kilos. No anesthesia. Intrapleural pressure readings after injection of atropine sulphate subcutaneously.

Jan. 9. Dose 6 mg.			Jan. 31. Dose 5 mg.		
	I.P.P. Left Chest in cm. H_2O	Mean I.P.P. in cm. H_2O		I.P.P. Left Chest in cm. H_2O	Mean I.P.P. in cm. H_2O
Normal	−0.8 to −5.8	−3.3	Normal	−2.0 to −6.0	−4.0
2 min. after atropine	−0.8 to −5.2	−3.0	3 min. after atropine	0.0 to −6.0	−3.0
5 min. after atropine	−0.8 to −5.2	−3.0	6 min. after atropine	+1.0 to −7.0	−3.0
8 min. after atropine	+0.6 to −4.8	−2.1	10 min. after atropine	−1.0 to −6.6	−3.8
11 min. after atropine	+2.8 to −4.8	−1.0	14 min. after atropine	−1.4 to −6.0	−3.7
13 min. after atropine	+5.2 to −2.8	+1.2	17 min. after atropine	−0.6 to −6.6	−3.6
15 min. after atropine	+5.2 to −2.8	+1.2	20 min. after atropine	+1.4 to −6.0	−2.3
			23 min. after atropine	+0.6 to −5.0	−2.2

other factors concerned in the various types of respiration, some of which will be discussed later.

Bronchoconstricting Drugs.[17]—Physostigmine (eserine) salicylate and pilocarpine nitrate cause bronchoconstriction by stimulating the postganglionic fibers of the craniosacral autonomics. Following the administration of these drugs, we found that the intrapleural pressure

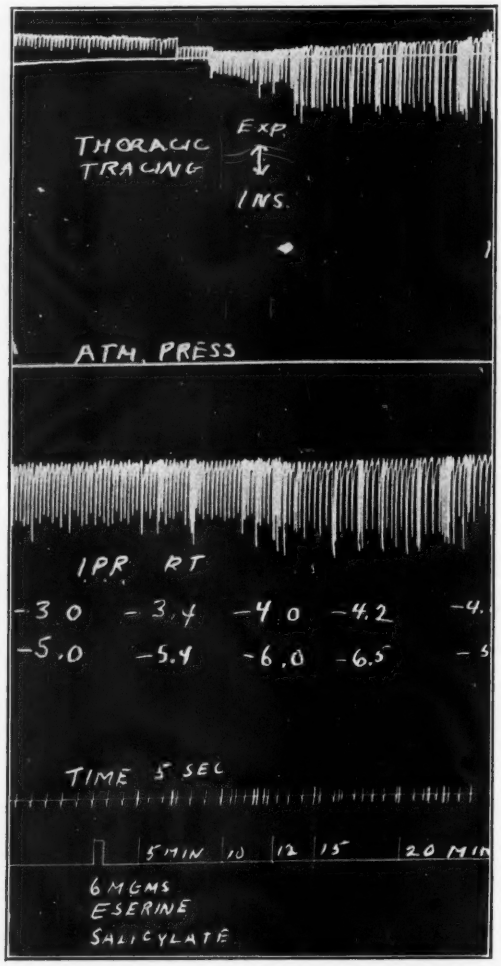

Fig. 5.—Kymographic record of intrapleural pressure and thoracic girth in a dog under sodium amytal anesthesia following injection of eserine. Upper tracing is respiratory curve; increase in the length of the down stroke indicates increase in girth of chest. Lower tracing is of intrapleural pressure (I. P. P.). Intrapleural figures are indicated in centimeters of water.

became more negative as is illustrated in Fig. 4.* It was found also that the mean thoracic girth increased (Fig. 5). In other words, in this instance respiration is taking place with the chest relatively in the inspiratory position.

In these experiments the effect of the anesthetic agent on intrapleural pressure was negligible. So far we have disregarded the possible effects of the bronchodilating and bronchoconstricting drugs on the heart and circulation, which in turn may affect the intrapleural pressure.†

TABLE IV

Effect on intrapleural pressure and thoracic girth of the subcutaneous injection of 6 mg. atropine sulphate in a 21 kilo dog anesthetized with sodium amytal.

	Intrapleural Pressure in cm. H_2		Thoracic Girth	
	Inspiration	Expiration	Inspiration	Expiration
			cm.	cm.
Normal	−2.8	0.0	25.2	25.1
5 min. after drug	−2.1	+3.0	25.0	24.8
10 min. after drug	−2.1	+7.0	24.8	24.5
15 min. after drug	−2.0	+8.0	24.6	24.4

EFFECTS OF GASES

Carbon Dioxide.[18]—The effects of the inhalation of carbon dioxide on the intrapleural pressure were studied by administering the gas either through a mask (by which method there was no control of the concentration) or through a tracheal cannula. Various concentrations of the gas were used with admixtures of oxygen.

TABLE V

Effects of the inhalation of 10% CO_2 in oxygen on intrapleural pressure and thoracic girth in a 17 kilo dog lightly anesthetized with sodium amytal.

	Intrapleural Pressure in cm. H_2O		Thoracic Girth	
	Inspiration	Expiration	Inspiration	Expiration
			cm.	cm.
Normal	−4.5	+0.5	54.6	54.2
CO_2 inhaled	−8.0	+0.5	55.3	54.2
CO_2 off	−6.5	+0.5	55.0	54.2

In all instances, in the intact animal, it was found that there was a gradual development of a more negative intrapleural pressure than exists normally. This was accompanied by an increase in the mean thoracic girth. (Fig. 6, Table V.) The effect is similar to, but more rapid than, that noted after the administration of bronchoconstricting drugs. Brodie and Dixon[19] found increased resistance to the passage of air in a cat after carbon dioxide inhalations, and Einhoven[20] re-

*Since completion of this article, we find that this observation was made originally by C. F. Hoover, in 1922. (Tracheal and Bronchial Stenosis as Causes for Emphysema; Arch. Int. Med. **29**: 142-167, 1922.)

†Suggested by Dr. Emile Holman, Stanford University Medical School.

ported that carbon dioxide caused increased tonic constriction of the bronchi. From this, many investigators concluded that carbon dioxide inhalations cause bronchial constriction. On the other hand, by direct bronchoscopic observation, Brown[21] of our clinic noted that the administration of carbon dioxide through the bronchoscope caused bronchodilatation. Cannon, Linton, and Linton[22] found that muscle metabolites, of which an important one is carbon dioxide, stimulate the sympathetic system, which in turn causes bronchodilatation. Douglas and Haldane[23] concluded that with increased alveolar carbon dioxide tension there must be a dilatation of the air passages.

Fig. 6.—Kymographic record of intrapleural pressure and thoracic girth in a dog under sodium amytal anesthesia following the administration of different concentrations of carbon dioxide. Intrapleural pressure figures are indicated in centimeters of water.

It can be seen then that accompanying the hyperpnea of carbon dioxide inhalations are two mechanisms: first bronchodilatation which facilitates the exchange of air, and second an increase in the mean thoracic girth, more negative intrapleural pressure, and greater distention of the lungs, all of which increase the respiratory exchange.

These findings of the effects of carbon dioxide may explain in part the effects of bronchoconstricting and bronchodilating drugs on the intrapleural pressure and thoracic girth. Bronchoconstriction, by causing increased resistance to the passage of air into and out of the

alveoli, may be expected to be accompanied by a gradually increasing carbon dioxide tension in the alveoli and blood. Davies, Haldane, and Priestly[24] showed that cotton-wool resistance to nasal breathing definitely increased the alveolar carbon dioxide percentage, and Meakins[25] found carbon dioxide retention in paroxysmal asthma.

We have shown that carbon dioxide inhalations, which cause increased carbon dioxide tension in the alveoli and blood, are accompanied by an increase in the mean thoracic girth and by a more negative intrapleural pressure. Therefore we may postulate that the effects of bronchoconstricting and bronchodilating drugs on intrapleural pressure are mediated by a sequence of events, causally related, as follows:[25]

Bronchoconstriction	Bronchodilatation
↓	↓
Increased alveolar and blood carbon dioxide tension	Diminished alveolar and blood carbon dioxide tension
↓	↓
Increased inspiratory muscular activity	Lessened inspiratory muscular activity
↓	↓
Increased average thoracic size	Reduced average thoracic girth
↓	↓
More negative intrapleural pressure	More positive intrapleural pressure

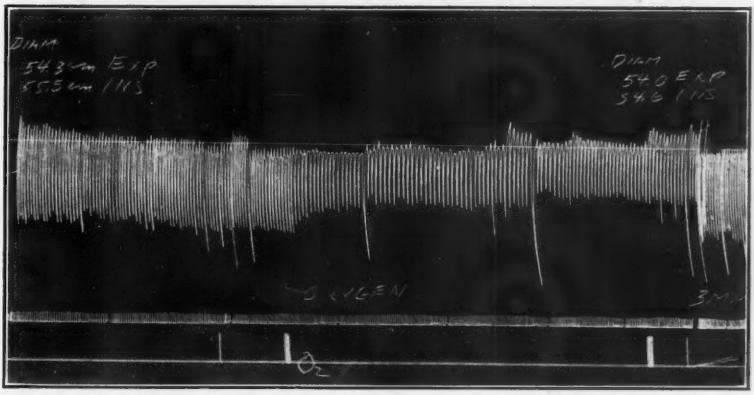

Fig. 7.—Kymographic record of the intrapleural pressure in a dog under sodium amytal anesthesia following the administration of oxygen. Horizontal line indicates atmospheric pressure.

While our experimental evidence presented in this and previous reports does not prove that a causal relationship exists between the sequence of events as listed in these schema, we feel that it is sufficient to establish a working theory in the absence of any significant evidence otherwise.

In all the experiments concerning the effects of gases (carbon dioxide, oxygen, anoxemia) the animals were under sodium amytal anes-

thesia, which is known to affect the respiratory mechanism. The experiments are to be repeated under other types of anesthesia.

Oxygen.—Upon the administration of oxygen through a mask, it was found in the majority of instances that the mean intrapleural pressure became slightly more positive (Fig. 7). In some instances there was apparently no effect, but in no case did the intrapleural pressure decrease (become more negative).

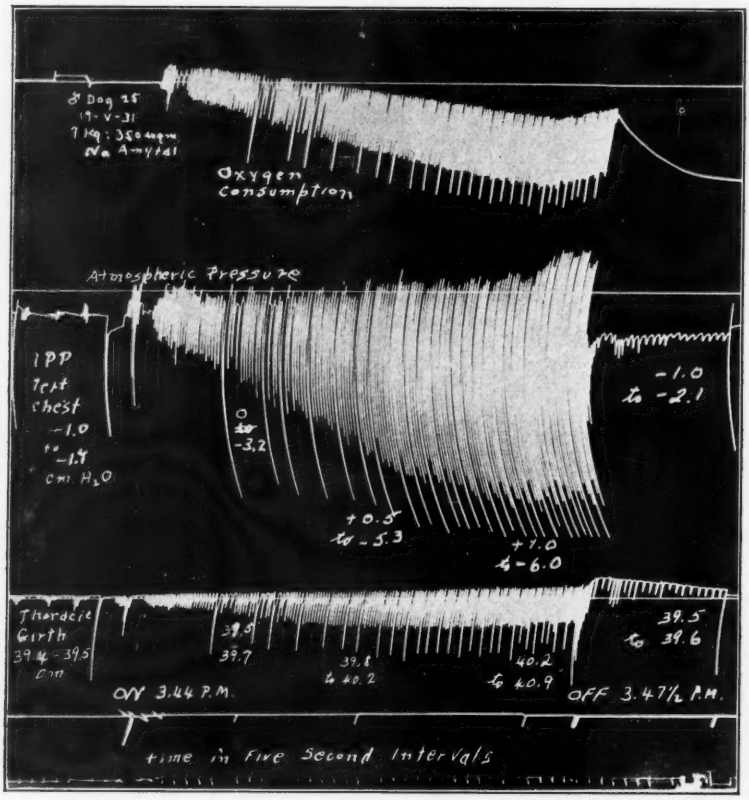

Fig. 8.—Kymographic record of intrapleural pressure and thoracic girth in a dog under sodium amytal anesthesia showing the effects of anoxemia. The upper tracing indicates the gradual consumption of oxygen from the Grogh's spirometer in the circuit of which is soda-lime to remove the carbon dioxide. Intrapleural pressure figures are in centimeters of water; thoracic girth figures are actual measurements in centimeters with a steel tape.

Anoxemia.—We investigated the effect on intrapleural pressure and thoracic girth of lack of oxygen without any increase in carbon dioxide tension. For this purpose we employed Krogh's spirometer of two liter capacity with soda-lime so placed in the respiratory circuit that there was no interference with breathing.

By placing a marker on the spirometer, oxygen consumption was recorded (Fig. 8). As the oxygen tension of the inspired air decreased the intrapleural pressure became more and more negative and the mean girth of the chest increased steadily. That is, the animal tended more and more to breathe in the inspiratory position. This is well illustrated in Fig. 8. In other words, the effect is very similar to that produced by carbon dioxide inhalation, but much more marked.

COMMENT

The clinical applications of the foregoing findings are complicated. The effects following carbon dioxide inhalations help rationalize its use as a therapeutic agent, especially in combating postoperative pulmonary complications. Henderson[27] suggested its use to combat "acapnia" during and following any surgical procedure. Since the recognition of the frequency of atelectasis as a postoperative pulmonary complication,[28] carbon dioxide inhalations have been found helpful in that they cause hyperpnea thereby increasing the expansion of the lungs as well as making the patient cough frequently, thus expelling pulmonary secretion. In addition, we have shown that carbon dioxide causes bronchial dilatation, increase of thoracic girth, and increased negative intrapleural pressure, all of which combat the tendency to localized and general collapse of the pulmonary tissue.

Physiologically it would seem that these effects of carbon dioxide inhalations increase the respiratory exchange and pulmonary circulation. Wright[29] states: "During inspiration the negative pressure in the thorax is increased. This pulls on the walls of the thin capillaries and venules which surround the alveoli of the lung and increases the lumen of these vessels. One-eighteenth of the blood volume can be accommodated in the pulmonary bed during expiration and one-twelfth during inspiration." When there is increased carbon dioxide retention in the alveoli and blood, as in exercise, these factors help maintain a good respiratory exchange.

In asthma, on the other hand, there is also an increased carbon dioxide tension during the attacks, and respiration takes place in the inspiratory position.[30] But expiration is impeded and for that reason respiratory exchange is diminished. The increased negative intrapleural pressure in addition to the obstructed, prolonged expiration may afford a partial explanation of the emphysema noted in chronic asthmatics.

The increased negative pressure and increased thoracic girth following anoxemia may, in a similar fashion, help explain the emphysema sometimes noted in those long exposed to rarefied atmospheres. When emphysema occurs, the lungs lose their elasticity, and the chest girth is permanently increased. Then the intrapleural pressure, which is dependent upon the elastic recoil, may become more positive, as was

found by Ameuille.[31] Even though increased carbon dioxide tension is present, the chest cannot be expanded further.

A similar type of reasoning may be followed in the consideration of patients with cardiac decompensation. They probably have both increased carbon dioxide tension and oxygen want, which tend to increase the respiratory exchange and cause respiration to take place in the inspiratory position. To verify this, we measured the diameter of the thorax at ordinary inspiration of four such patients, and compared these figures with their maximum inspiratory effort (Tables VI, VII). It can be seen from these figures that respiration in these patients is taking place in the extreme inspiratory position. Increased carbon dioxide tension and oxygen want cannot produce any further expansion of the chest.

TABLE VI

Measurements of thoracic girth of patients with cardiac congestive failure.

DIAMETERS OF THE THORAX IN THE UPRIGHT POSITION

Case	Age	Diagnosis	Normal Expir. Diam.	Normal Inspir. Diam.	Maximum Expir. Diam.	Maximum Inspir. Diam.	Diff. Between Normal and Maximum Inspir. Diam.
			cm.	cm.	cm.	cm.	cm.
1	54	Aortic Insuff.	92.7	93.1	91.6	93.2	0.1
2	54	Aortic Insuff.	94.9	95.1	93.2	96.0	0.9
3	5	Rheumatic Myocarditis	55.7	56.0	55.0	56.0	0.0
4	40	Mitral Stenosis	86.3	87.1	85.7	87.3	0.2

These figures are the average of three measurements at the nipple line.

TABLE VII

Measurements of thoracic girth of patients with cardiac congestive failure.

DIAMETERS OF THORAX IN CARDIACS IN THE SUPINE POSITION

Case	Normal Expir. Diam.	Normal Inspir. Diam.	Maximum Expir. Diam.	Maximum Inspir. Diam.	Diff. Between Normal and Maximum
	cm.	cm.	cm.	cm.	cm.
1	91.6	91.7	98.7	93.0	1.3
2	92.8	93.0	90.0	94.0	1.0
3	54.3	54.5	54.0	55.0	0.5
4	85.8	86.5	84.8	87.3	0.8

These figures are the average of three measurements at the nipple line.

EFFECTS OF PNEUMOTHORAX

Records were obtained from dogs with open and closed pneumothorax to confirm what has already been shown by others.

Fig. 9 illustrates the effect on intrapleural pressure and thoracic girth of repeated injections of air into the chest. When small amounts of air are injected into the pleural cavity, the intrapleural pressure rises in direct proportion to the amounts of air injected. This is true of the intrapleural pressure in both pleural cavities (Figs. 9, 10). At

the same time the thorax compensates by expanding, that is, by increasing its thoracic girth. This mechanism has been discussed well by Graham.[12] Conversely, when air is withdrawn, the intrapleural pressure tends to return to its subatmospheric level and the thoracic girth again decreases (Fig. 9).

Since both cavities are encroached upon by the pneumothorax, the respiratory exchange is greatly decreased. This is illustrated in Fig. 11. The upper tracing gives a rough measurement of the amount of

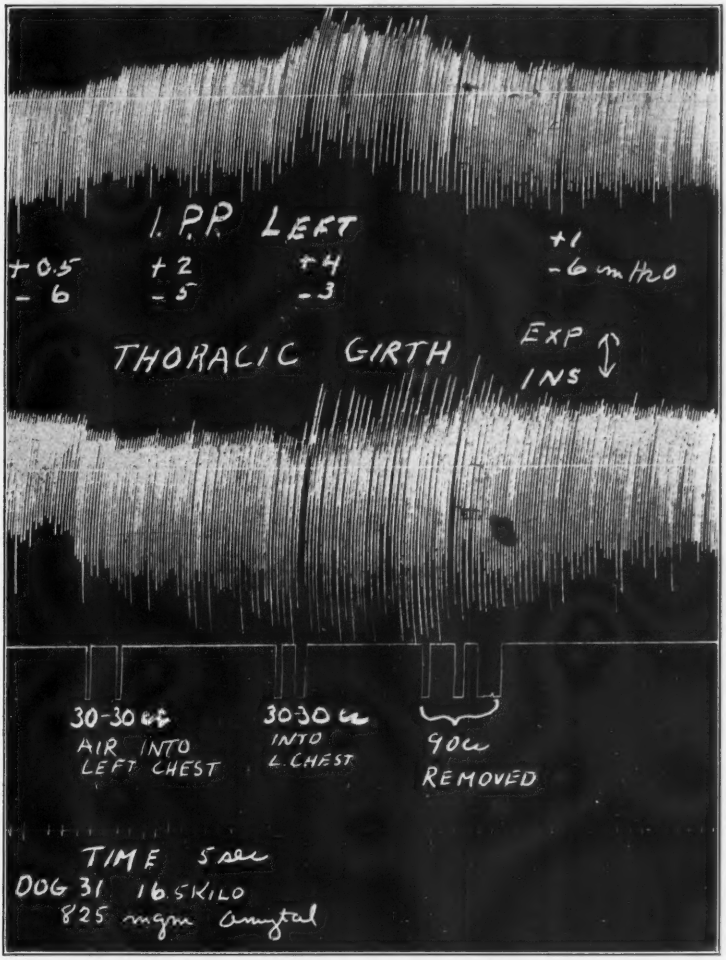

Fig. 9.—Kymographic record of intrapleural pressure and thoracic girth in a dog under sodium amytal anesthesia showing the effects of the injection and withdrawal of air in the pleural cavity. In this instance the intrapleural pressure tracing is of the same side of the chest into which the air was injected. Figures indicate intrapleural pressure in centimeters of water.

inspired air. It can be seen that when the thoracic wound is opened the intrapleural pressure is positive and the respiratory exchange is practically nil, and that when the pneumothorax wound is closed the pressure again becomes negative and the respiratory exchange is almost normal. The importance of closing the pneumothorax at the end of expiration when there is the least amount of air in the pleural cavity is illustrated in Fig. 12. If the wound is closed at the end of inspiration the intrapleural pressure is much more positive and the

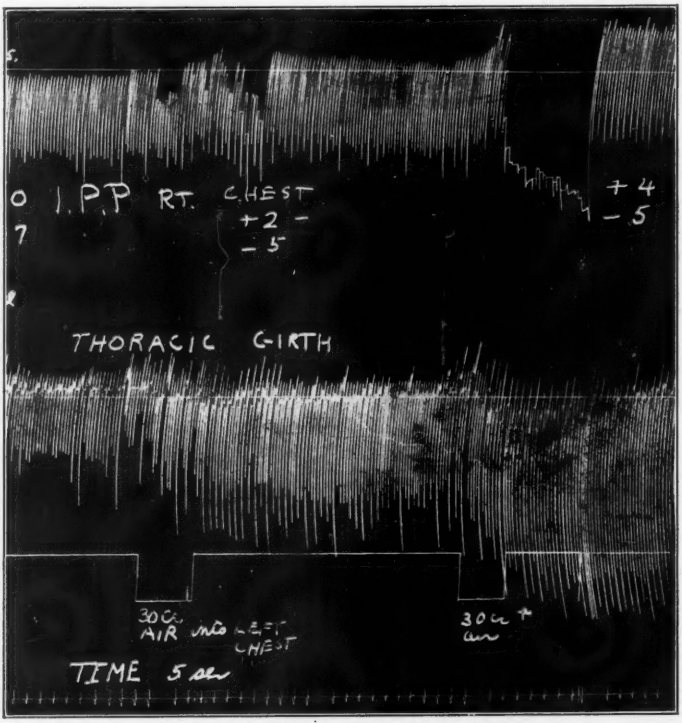

Fig. 10.—Same as Fig. 9 except that in this instance intrapleural pressure is taken on the side of the chest opposite to that into which air is injected.

amount of inspired air is greatly decreased, while if the pneumothorax is closed at the height of expiration the intrapleural pressure is largely negative and the inspired air almost returns to normal in amount. These tracings are shown merely to illustrate graphically facts that are understood by all thoracic surgeons and that have been well explained by Graham.[12]

EFFECTS OF SURGICAL PROCEDURES

Little is known about the relation between intrapleural pressure and intraperitoneal pressure. Most of the work in intraperitoneal

pressure has been unsatisfactory. Clark[32] introduced a cannula into the trachea of animals and connected a manometer to the cannula. Upon opening the abdomen he found no change in the intrapleural pressure. He therefore concluded that intraperitoneal pressure probably had little or no effect on intrapleural pressure. However, Overholt[33] concludes, "The fact that pressure within the peritoneal cavity decreases simultaneously with decrease in intrapleural pressure (during inspiration) suggests a close relationship between the abdominal cavity

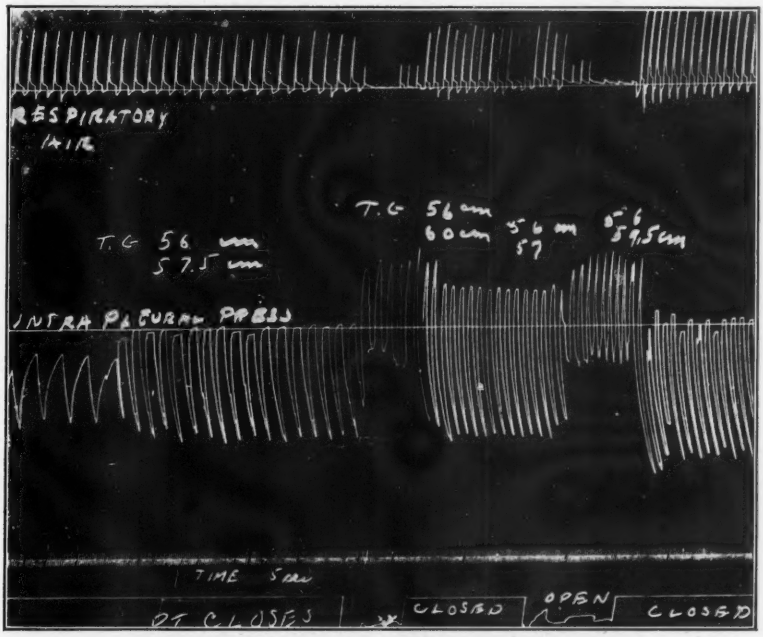

Fig. 11.—Kymographic record of the respiratory exchange and intrapleural pressure in a dog under sodium amytal anesthesia showing the effects of open and closed pneumothorax. The upper tracing gives a rough estimate of the amount of inspired air with each respiration, and was obtained by having a "Y" tube connected to a tracheal cannula. One end of the "Y" tube was connected to a tambour and the other end left open. T.G. figures indicate thoracic girth measurements. P.T. = pneumothorax.

and respiration. It also suggests the possibility of interference with pressure variations above the diaphragm by opening of the sealed cavity below."

In a preliminary report[34] we noted that various surgical procedures, such as the administration of ether, opening the abdomen, etc., produce changes in the intrapleural pressure. At this time we should like to report further data on various surgical procedures.

It is not uncommon for some member of an operating team when he becomes fatigued, to lean on the patient's abdomen. Fig. 13 illustrates the effects on intrapleural pressure and thoracic girth of pressure on

the abdomen. As might be expected, the intrapleural pressure immediately becomes more positive while the mean thoracic girth is markedly increased. This undoubtedly interferes with the normal respiratory exchange.

The effects of the administration of an anesthetic, such as ether, are interesting. During the induction, the intrapleural pressure is markedly increased toward both the positive and negative sides, with expiration and inspiration respectively, accompanying the increased respiratory excursion. As deep anesthesia sets in, the intrapleural pressure be-

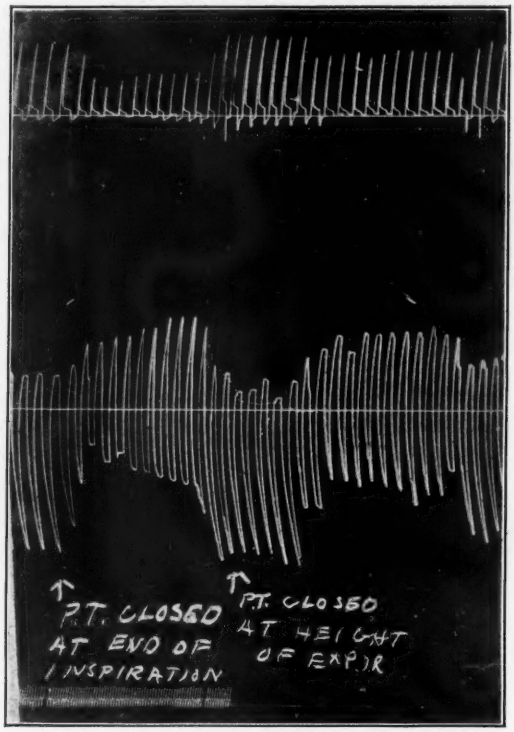

Fig. 12.—Kymographic record of the respiratory exchange and intrapleural pressure in a dog under sodium amytal anesthesia showing the effects of closing the pneumothorax at the end of inspiration and at the end of expiration. Upper tracing is a rough estimate of respiratory exchange, and lower tracing is of intrapleural pressure.

comes more positive than normal so that finally, during expiration, it is well above atmospheric pressure, and during inspiration, although still subatmospheric, it is much less so than before the anesthetic was applied. Coincidently the mean thoracic girth decreases markedly. After the anesthetic is removed the intrapleural pressure and thoracic girth tend to return to their preanesthetic levels (Fig. 14).

When the abdomen is opened (Fig. 15) the intrapleural pressure immediately rises while the blood pressure falls. At the same time the mean thoracic girth increases. The effect, then, is similar to that obtained by pressure on the abdomen, and tends to impede normal respiration.

Manipulating the organs within the abdomen apparently has little effect on the intrapleural pressure or blood pressure (Fig. 16). This again illustrates the importance of keeping the viscera in the peri-

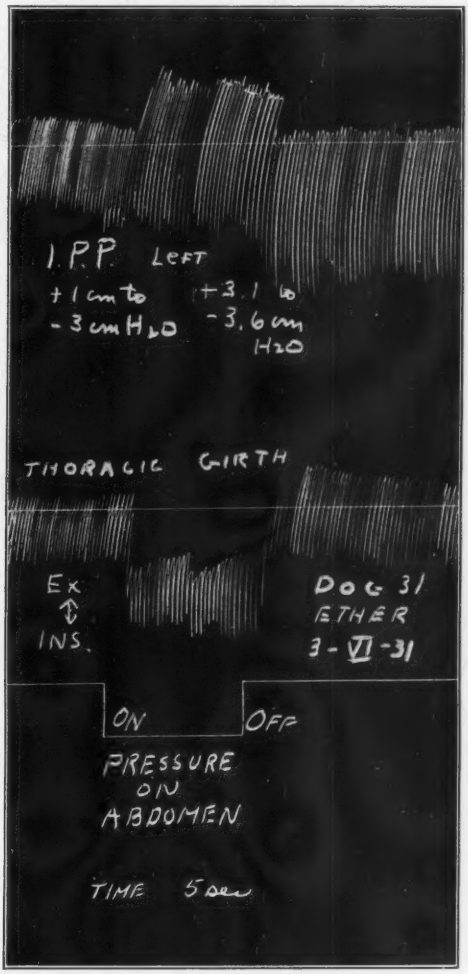

Fig. 13.—Kymographic record of intrapleural pressure and thoracic girth in a dog under ether anesthesia showing effects of pressure on the abdomen. I.P.P. figures indicate intrapleural pressure in centimeters of water.

toneal cavity whenever possible. When the viscera are removed from the abdomen, the intrapleural pressure immediately becomes more positive and the mean thoracic girth increases (Fig. 17). Traction on the various organs or the mesentery (Fig. 18), as is well known, causes marked changes in respiratory physiology. The changes are all of similar type and are well illustrated in Fig. 19, which is a graph showing the effects of traction on the stomach such as may occur during any surgical procedure. It can be seen that there is marked increase in the intrapleural pressure accompanied by a moderate fall

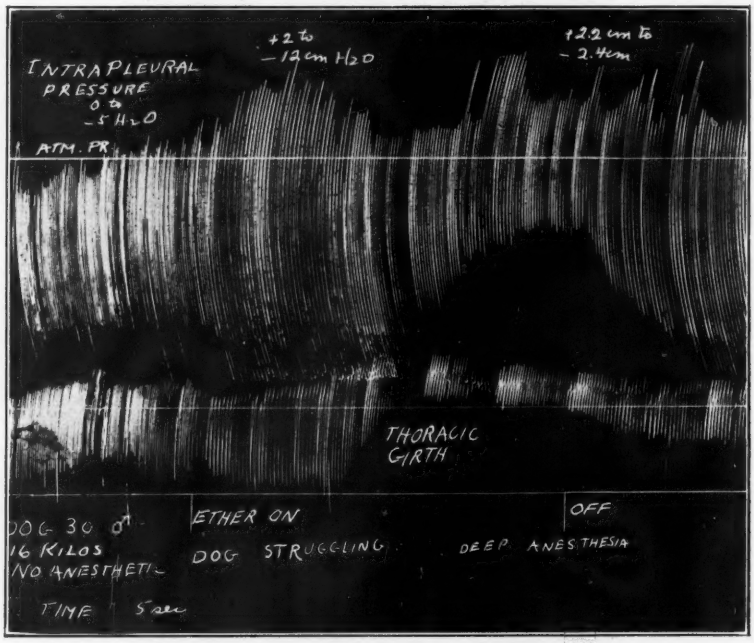

Fig. 14.—Kymographic record of intrapleural pressure and thoracic girth in a dog, showing effects of ether. Intrapleural figures indicate actual pressure in centimeters of water.

in blood pressure. Surgical procedures in general, then, tend to cause an increase in intrapleural pressure and, with the exception of ether anesthesia, an increase in the mean thoracic girth. This condition probably persists postoperatively as shown by Muller, Overholt, and Pendergrass[35] who, both before and after upper abdominal surgery, actually measured the thoracic girth of patients and noted in fifteen cases an average increase of about 2.5 cm. on the first postoperative day. They noted also that there was a significant rise in the diaphragm and that its movement was retarded. These facts are no doubt related to the increased intrapleural pressure. If we assume

that the increase in intrapleural pressure (more positive pressure) also persists postoperatively, it will explain partially the markedly decreased vital capacity that these people have. All these factors indicate that the pulmonary mechanism is placed under a very real

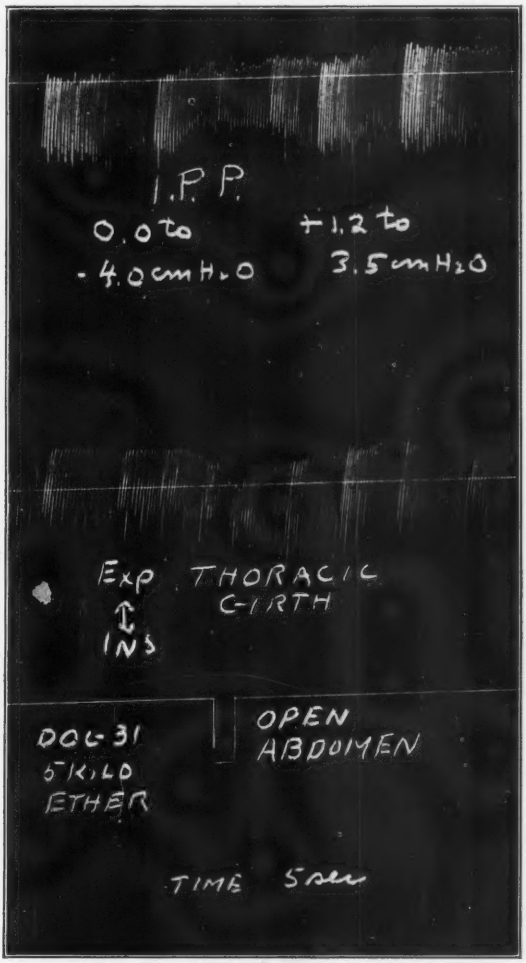

Fig. 15.—Kymographic record of intrapleural pressure and thoracic girth in a dog under ether anesthesia showing effects of opening the abdomen.

handicap in many surgical procedures. We may point out again that carbon dioxide inhalations tend to combat most of the effects of the operative procedures mentioned by increasing the negative pressure and by causing bronchodilatation and hyperpnea.

If we consider the effects of the various procedures on intrapleural pressure and thoracic girth, they seem to fall into two general groups:

Group I (drugs and gases) causes either increase in intrapleural pressure accompanied by a decrease in the mean thoracic girth, or decrease in intrapleural pressure (more negative) accompanied by increase in mean thoracic girth.

Group II (mechanical means such as pressure on the abdomen, sur-

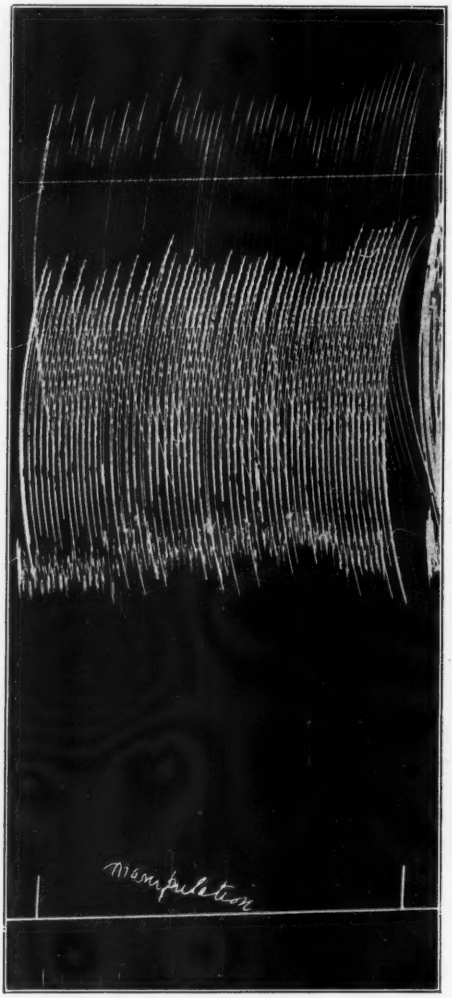

Fig. 16.—Kymographic record of intrapleural pressure of both chests and the blood pressure in a dog under ether anesthesia showing that manipulation of viscera within the abdomen had little effect. Blood pressure tracing can be seen in lower part of record.

gical procedures, and pneumothorax) causes increased intrapleural pressure accompanied by increase in the mean thoracic girth, or decrease in intrapleural pressure accompanied by decrease in thoracic girth.

In other words, in the case of operative procedures we seem to have a purely physical mechanism, while the effects of the drugs seem to depend on a mechanism mediated by reflex effects resulting from

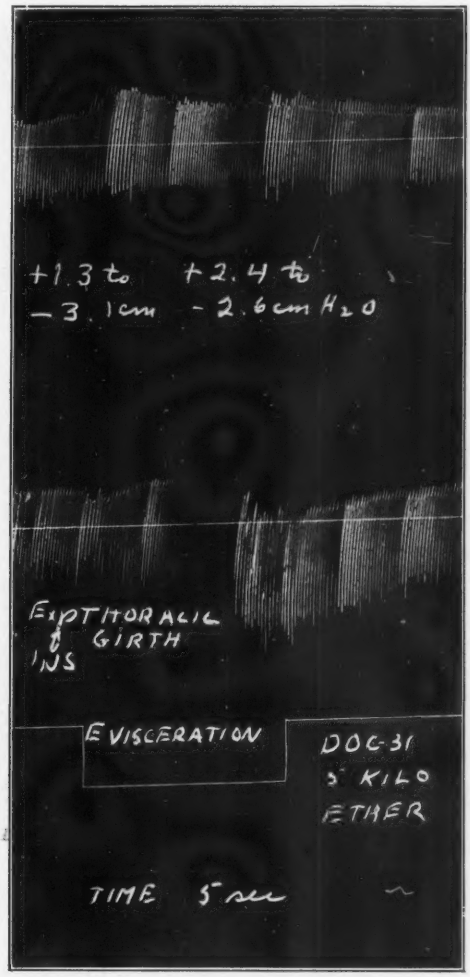

Fig. 17.—Kymographic record of intrapleural pressure and thoracic girth in a dog under ether anesthesia showing effects of evisceration. Upper tracing is of intrapleural pressure and figures indicate actual measurements in centimeters of water. Lower tracing is of thoracic girth.

pharmacological action. In the case of pneumothorax, the changes in thoracic girth are compensatory to the decreased vital capacity.

SUMMARY

Normal intrapleural pressure is subatmospheric. The administration of bronchodilating drugs, such as epinephrin and atropine, is followed by an increase in the mean intrapleural pressure and decrease in the mean thoracic girth. After bronchoconstricting drugs,

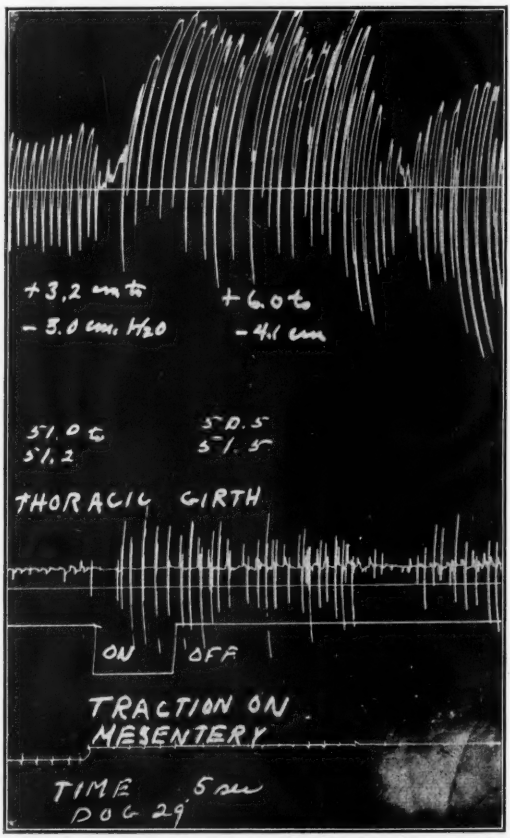

Fig. 18.—Kymographic record of intrapleural pressure and thoracic girth in a dog under ether anesthesia showing the effects of traction on the mesentery.

such as pilocarpine and eserine, there is a decrease in the mean intrapleural pressure and an increase in the mean thoracic girth. Carbon dioxide inhalations are followed by a more negative intrapleural pressure than exists normally, accompanied by an increase in the mean thoracic girth. The changes in intrapleural pressure following the

administration of bronchoconstricting and bronchodilating drugs may be the result of the increased or decreased resistance to the passage of air through the bronchi, or may be the result of changes in the alveolar and blood carbon dioxide tension which in turn cause changes in thoracic girth and intrapleural pressure.

Our observations of the effects on intrapleural pressure of carbon dioxide inhalations lend further support to its use as a prophylactic and therapeutic agent in postoperative pulmonary complications.

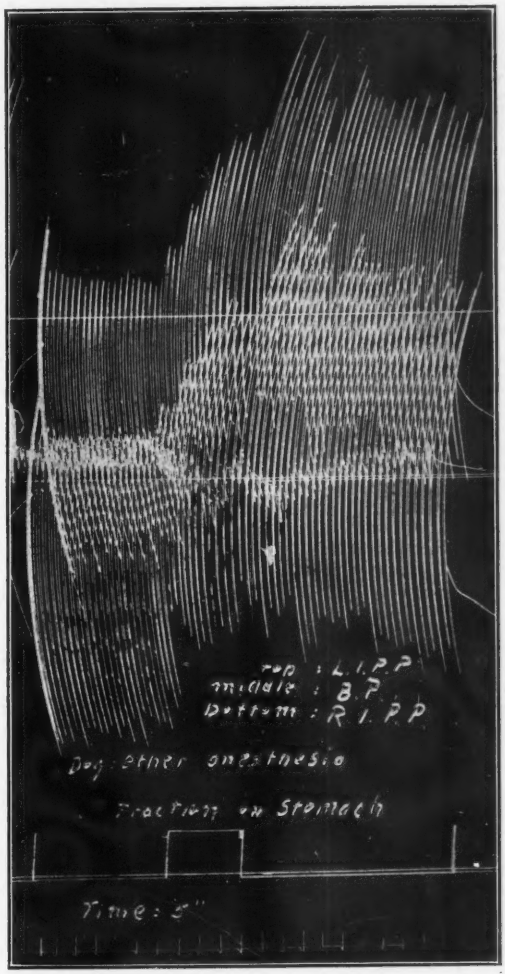

Fig. 19.—Kymographic record of intrapleural pressure in both chests and the blood pressure in a dog under ether anesthesia showing the effects of traction on the stomach. Blood pressure tracing can be seen superimposed on the intrapleural pressure tracings.

Anoxemia is followed by a steadily decreasing intrapleural pressure which is coincident with increase in the mean thoracic girth. The effect is even more marked than that following carbon dioxide inhalations.

Some of the effects of pneumothorax are illustrated graphically.

Experimental surgical procedures, such as pressure on the abdomen, administration of ether, opening of the abdomen, traction on the stomach, and evisceration, were followed by an increase in the intrapleural pressure and an increase in the mean thoracic girth. Such procedures place the respiratory mechanism under a handicap, but the untoward effects may be counteracted in part by carbon dioxide inhalations. Other clinical applications of these studies are discussed.

We desire to thank Professor Chauncey D. Leake for his interest and aid in this work.

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DISCUSSION

DR. GEORGE P. MULLER.—Dr. Brill and Dr. Brunn are to be congratulated for this fine piece of work. If I may say so, Dr. Brill has carried on still further certain findings which Overholt got in our Clinic. The work of these men and some others of our Association has done much to solve the problem of postoperative pulmonary complication. Certainly, we have learned that the embolic factor is not the correct one in the majority of cases. The next step for study and experiment is the development of some means by which such complications can be avoided.

DR. BRILL.—I would like to add one fact which was not brought out in the paper. In discussing the effects of drugs on intrapleural pressure, I did not mention that we did not consider the effects of these drugs on the blood pressure or circulation.

Dr. Holman had pointed out previously that the effects of these drugs or gases on the circulation and on blood pressure may secondarily cause changes in the intrapleural pressure. We hope to study these factors at another time.

BRONCHIAL STENOSIS*

(*Continued from p. 213*)

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II.

PHYSIOLOGY

Obstruction of the air passages has been studied experimentally by Traube[5] (1846 and 1871) and Lichtheim[6] (1878) whose classical researches are a base upon which most later investigators have built. Bohr[35] and Hasselbach,[36] both of Copenhagen, Volhardt,[37] Schall,[38] Siebeck[39] and Morawitz,[40] Hoover[11] and Bruns[41] added valuable data; more recently Nissen[12] of Sauerbruch's clinic has published an exhaustive series of researches on the effects of partial and complete stenosis and Sulger[42] has contributed to our knowledge of the influence of stenosis on circulation. The painstaking studies of Coryllos[43] and his coworkers, of van Allen,[44] Lee[45] and Brunn[46] and many others on acute obstruction do not concern us here. The conclusions of many investigators are at variance, not only with each other, but with clinical phenomena as observed at the bedside. The discrepancy is to be sought in the complexity of the factors inducing stenotic changes. Perfect clarity and coincidence of opinion are difficult to reach.

Lichtheim,[6] studying the cause of atelectasis, plugged the bronchi of rabbits with laminaria tents. He observed the massive collapse and dilatation of the contralateral lung that ensued. For three or four hours after operation his animals were dyspneic and their breathing was forceful and rapid; this dyspnea receded, but after twenty-four to forty-eight hours many of them were seized by a sudden violent spasm, knocked the covers off their cages and were found dead on the floor. He was unable satisfactorily to explain the spasm. Occasionally the contralateral lung burst with production of pneumothorax the cause of which was also obscure. Rabbits with incomplete stenosis survived, as did those in which the laminaria tent had perforated a bronchus, causing homolateral pneumothorax. Lichtheim was unable to control the site and degree of obstruction when he used tents; it is not unlikely that the tents as they swelled obstructed other bronchi than the one into which they originally had been introduced. In some animals he ligated the bronchi instead of plugging them; these rabbits lived from four to six weeks and finally succumbed to empyema (the experiments were done before the days of asepsis). Of this series he writes,

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Read before the Thirteenth Annual Meeting of the American Association for Thoracic Surgery, Philadelphia, May 19, 1930.

"Either the whole bronchial tree was very greatly dilated and filled with white viscid pus, and the pulmonary parenchyma itself was reduced to a minimum forming a fibrous abscess wall scarcely a millimeter thick which reminded one of pyelonephrosis, or the bronchiectasis was of a less degree, the bronchi lying imbedded in atelectatic parenchyma." A series of exact determinations showed that the cause of atelectasis lay in diffusion of air through the alveolar wall and absorption by the blood stream.

Nissen[12] worked with 4 dogs, 6 rabbits, and 3 cats. He worked under differential pressure, inflated the lung, isolated the bronchus, crushed it and tied it off. He tied the right or left major bronchus in most of the rabbits and cats; in the dogs one of the lobar bronchi. The obstructed part of the lung became atelectatic in two to four hours and filled with secretion in three to four days. The secretion remained aseptic; indeed, he found that the ligated lobes were not affected during an epidemic of distemper from which his animals suffered. Animals killed after three months showed contraction and carnefication of the ligated parts of the lung with dilatation of the bronchi. Both Lichtheim and Nissen produced sudden complete obstruction of a bronchus, comparable to that of a foreign body; many other investigators have busied themselves with obstruction of the trachea. It was evident from the outset of these investigations that when the air passages are constricted as a whole, obstruction to inspiration (a pure suction-valve stenosis) and obstruction to expiration (a pure pressure-valve stenosis) must have utterly different, perhaps diametrically opposite, effects, and that neither will permit, a priori, of deduction of the effects of a block obstructing both inspiration and expiration simultaneously. This distinction was noted by the first observers. Cohnheim[47] in 1882 cited a pedunculated tumor lying above the glottis as an example of purely inspiratory stenosis, and a similar tumor below the glottis as an expiratory stenosis. Bohr,[35] Hasselbach,[36] Bruns,[41] Hoover,[11] Forschbach and Bittorf[48] and others made spirometeric measurements on human subjects whose respiration they obstructed with suction and pressure valves. More recently Pfanner[49] attempted to draw clinical deductions from a series of animal experiments and to place all cases of obstruction in one or the other category. His experiments are unconvincing because they lack quantitative data and because his valves were so close that his animals died in the first twenty-four hours. Purely inspiratory and purely expiratory obstruction are after all rare; most stenoses affect both respiratory phases; which elements preponderate, suction or pressure, when and why, are still not perfectly clear.

The results of most artificial tracheal obstructions in animals and man concur with bedside observations. Partial stenosis of considerable degree causes at first a stage known as inspiratory dyspnea or

stridor in which inspiration is prolonged, deeper and more forceful. The intercostal spaces are retracted, intrapleural pressure is lowered, and intrapleural manometric oscillation between inspiration and expiration is increased. Both time and depth of expiration are affected less or not at all, so that the tidal air is increased through an inspiratory increase, the residual air remaining constant or being increased, but never decreased (Hasselbach); i.e., the lung is dilated by active inspiration. If stenosis is excessive, or if muscular exertion increases the need for oxygen this stage soon goes over into one in which breathing is no longer slow and deep, but rapid and shallow; the lungs are dilated, intrapleural pressure approaches zero and death is likely to supervene.

Origin and immediate cause of the inspiratory dyspnea which characterizes tracheal stenosis are difficult to determine, for changes in the blood gases, in the O_2 and CO_2 content are not primarily at fault, as Bohr,[35] Haldane,[50] Henderson,[51] et al. first assumed. The normal type of breathing changes immediately stenosis is produced, long before changes in the blood gases are perceptible; the characteristic stenotic type disappears after vagotomy (Nissen[12]) and in diabetic coma. The stenotic change, therefore, is induced by a nervous, not a chemical mechanism, although it may be maintained by a chemical one. Higher cortical centers, the functions of which elude exact determination probably play a considerable rôle.

Finally there is no doubt that the effects of stenosis do not vary proportionately to its degree, but that quite suddenly an upset of respiratory compensation occurs which still awaits elucidation.

From the foregoing one may gather that moderate obstruction of the air passages influences mainly inspiration. A simple trial will show that if the nostrils are gradually closed resistance to inspiration comes to one's notice first, while expiration is still accomplished without effort; inspiration is still the active, expiration the passive phase of the respiratory act. A *slight* obstruction acting equally against inspiration and expiration increases inspiratory negative pressure but influences expiratory positive pressure to a lesser degree.

In order that the same amount of air be exchanged in each minute the more difficult inspiration is prolonged, is deeper and more forceful, while expiration proceeds relatively unimpaired. The beginning of expiration is, if anything, easier than normal, for a portion of the increased inspiratory negative pressure is carried over into the expiratory phase, and continuing longer in this phase than it normally should, aids the beginning, at least, of the expiratory act. This slowing of respiration is manifest clinically in ordinary compensated moderate tracheal and bilateral bronchial obstruction. Inspiration, though impeded, succeeds through greater depth and forcefulness, in accomplishing a normal interchange of air in a unit of time.

If the constriction is gradually narrowed so that not only inspiration but expiration requires appreciable effort or if during the stenosis respiratory demands are suddenly increased through muscular exertion or fever or a failing heart, then a notable change occurs. For the whole respiratory mechanism is set toward increasing its intake but not its output. Intake is active, output passive. This is true in quiet breathing and is still true in forced breathing, as after exertion. In nature the intake increases with increasing respiratory demands, but not correspondingly the output; more air enters the thorax; inspiration is deeper, more rapid, more forceful; expiration is relatively less influenced. Nature has provided means for overcoming inspiratory difficulties; against expiratory difficulties she stands ill-prepared. A trial at breathing against a pressure mask will show how tiring is disturbance of expiration.

Fig. 24.—Case 7. Tracheal stenosis. Graph of respiration. Prolonged inspiration both at rest and after exercise.

Innate deficiencies of the expiratory mechanism therefore lead to a vicious circle when expiration is interfered with; under conditions of stenosis in which the intake is increased but the output cannot be, an increasing amount of air must be entrapped in the lung behind the constriction. That this does occur was shown by Thornton and Pratt[52] experimentally and in a mechanical model; fluoroscopy and autopsy demonstrate it regularly. As retention of air increases a breakdown must occur, the cause of which is not clear, but which may lie in obstruction to return of peripheral blood to the thoracic veins (Sulger[42]). It seems not improbable that the appalling attacks of cyanosis, dyspnea and collapse observable at the bedside are due to the sequence: expiratory difficulty, acute dilatation of the lung, decompensation of the respiratory and circulatory mechanism and peripheral venous stasis. To restore equilibrium one of two things must happen: either intrapulmonary air pressure behind the constriction must increase to a point at which it overcomes the resistance to expiration, or collapse and a drop in blood pressure must release the constriction to a degree where breathing is again possible. Failing either, suffocation must occur.

These two forms of dyspnea: the constant, mainly inspiratory form with slow, deep inspiration, and the acute attack with rapid shallow breathing, a small fleeting pulse and collapse will be determined then, partly by the degree and site of obstruction, partly by resiliency of the lung, partly by complicating factors—competency of the circulation, dilatation of the right heart, presence of infection, etc., of which more later. Both forms occur and are observable at the bedside.

Other factors besides incompetency of the expiratory muscular arrangement increase the difficulty of continued respiratory effort. Circulatory complications play so decisive a rôle in the outcome of stenosis that it is seldom possible to determine whether responsibility for the terminal changes rests with the circulatory or respiratory apparatus. For in tracheobronchial stenosis the peripheral veins are regularly found dilated at autopsy and engorged with blood, the right heart is usually dilated and sometimes if there has been an attempt at compensation, hypertrophied.

Sulger[42] and Nissen[12] both showed that contrary to our previous notions pulmonary arterial pressure never rises in stenosis. In inspiratory stenosis it falls; in expiratory stenosis and in usual stenosis involving both inspiration and expiration it may remain unchanged, but in no form, nor in any degree of stenosis does it rise. It followed from their experiments that dilatation of the right heart is due to decreased pressure in the intrathoracic veins and increased suction to the right auricle. The right heart is fed more blood than its musculature is able to handle and it reacts with dilatation. Expiratory effort obstructs the flow in the great veins; the heart is not dilated but is inclined to run empty. Sulger found the right heart of dogs with expiratory dyspnea not enlarged but small and empty, the peripheral veins, however, were engorged. This agrees with some autoptic observations in emphysema. These experiments bring us closer to an understanding of the changes in the heart accompanying stenosis; dilatation in some stages of inspiratory dyspnea, diminution in size in expiratory dyspnea.

When the pulmonary veins are obstructed engorgement of pulmonary capillaries further impairs respiration; v. Basch[53] has shown that the lung is so stiffened hereby that there is little difference between inspiratory and expiratory air content. Tumors and vascular swellings that obstruct the tracheobronchial lumen will swell with pulmonary congestion and further increase the degree of obstruction. Explanation of the respiratory phenomena accompanying tracheal stenosis therefore is by no means simple. The difficulties to be overcome before a solution is reached may be apparent from the foregoing attempt at their exposition.

CASE 15.—A seaman and adventurer of sixty was admitted to the San Francisco Hospital on August 25, 1930. Four months previously he had begun to

complain of pain in the back and a cough which would come on in two or three paroxysms daily, making the pain much worse. This pain had come on quite suddenly on August 10, when he noticed on walking upstairs that he was very short of breath. Two days later this dyspnea became constant and he brought up a waterglassful of foamy sputum, "like peroxide."

On October 18 I found him markedly dyspneic, both on inspiration and expiration. The supraclavicular fossae were retracted on inspiration and he had an inspiratory crowing stridor. Expiration was markedly prolonged and difficult. He sat with his neck stretched, the head being bent forward with the chin up.

The right radial pulse was a little fuller than the left; the right fingers were clubbed. The blood Wassermann reaction was triple plus. Urine and blood analyses were normal.

The x-ray plates showed a displacement of the trachea to the left with extrinsic pressure on its right wall. Whether this was due to aneurysm of the innominate artery or to tumor was uncertain. The larynx was normal.

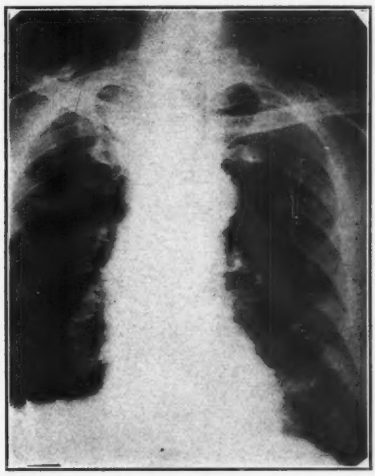


Fig. 25.—Case 15. Tracheal stenosis from probable aneurysm of innominate artery. Note indentation of right side of trachea.

Although the tracheal obstruction was complicated by an asthmatic and emphysematous component, expiration being prolonged and wheezy and the chest barrel-shaped as in true asthma, exercise caused a reversion in the type of respiration. After exercise inspiration was deeper and prolonged, but expiration greatly shortened. The change is documented in the accompanying tracing which Dr. van Deventer was kind enough to make. (Fig. 26-*A*.)*

PATHOLOGY

What are the ultimate anatomical results of stenosis? We know that increase in pressure proximal to constrictions of most hollow viscera leads to their gradual dilatation. If then a constriction affect-

*Mediastinal decompression was done, revealing an aneurysm of the innominate artery compressing the trachea. Dyspnea was immediately relieved. The man still has his aneurysm (Dec. 3, 1931).

ing expiration leads to an increase of intrapulmonary air pressure and entraps air in the bronchi, should it not dilate them? Dilatation of the lung and bronchi is determined by various factors. The thorax is rigid and its capacity limited; dilatation cannot proceed indefinitely. A certain increase in lung volume ("volumen auctum") is possible

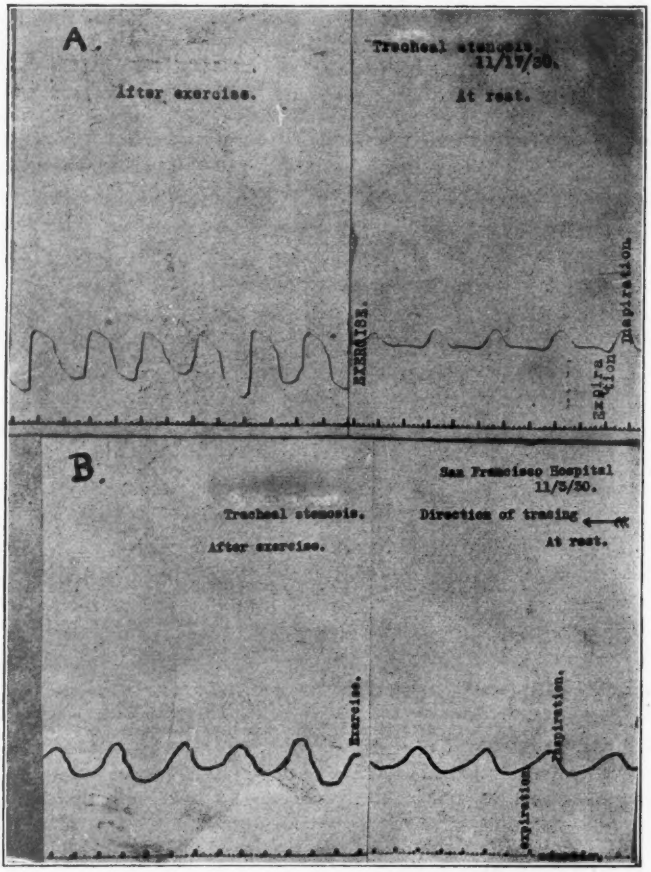


Fig. 26.—Case 15. Respiratory graph. Two types of respiratory change at rest and after exercise: A. Breathing more rapid, inspiration prolonged, deeper; expiration greatly shortened. B. Rate little changed.

and takes place in asthma and many high stenoses affecting both lungs at once, but when a localized portion of the bronchial tree is affected expansion of one portion takes place at the expense of others. Not only the site of obstruction but various other complicating factors determine the site and extent of dilatation. If the bronchioles are constricted dilatation of the alveoli and later emphysema result, the alveoli being not only dilated but fused into large air chambers. Why

this dilatation is not uniform and why some alveoli are blown out and fused into large bubbles while others are unaffected cannot be said. Again: which alveoli are dilated and which remain unaffected, the anatomical relation of the unaffected and the affected areas to the bronchioles, these are questions which, although Loeschke[54] has shed much light on them, still demand further study.

The problem grows more complicated in more central stenosis. It is true that when the constriction involves the larger bronchi the depending smaller ones are quite regularly found dilated and their ends ectatic, sometimes tubular, sometimes bottle-shaped. It is understandable that dilatation is more frequent in the lower more expansile and movable portions of the lung, but why it involves the bronchial system alone without producing emphysema at the same time is hard to explain. Similar irregularity occurs in other organs. Ureteral block produces a hydronephrosis with dilatation of the kidney pelvis but not of the tubules; constriction at the papilla of Vater causes dilatation of the common duct but not so regularly of the finer biliary tubules.

The inconstancy of dilatation is more understandable, for if it is true that mild constriction influences only inspiration and lowers rather than raises intrabronchial air pressure, then a mild constriction should not cause dilatation, but rather the contrary. Schall[38] found this to be true with a valve-mask mildly impeding inspiration. And again, if impairment of expiration with a consequent rise in intrabronchial pressure and retention of air in the lung is likely soon to be followed by circulatory decompensation and collapse, then of necessity the postulates favoring dilatation are unstable and can exist only within certain narrow limits.

As a matter of fact we do see tracheal and bronchial constriction without either bronchiectasis or emphysema; and even more frequently does dilatation affect only a circumscript portion of the bronchial twigs or the lung dependent from a constricted bronchus while other branches dependent from the same bronchus escape.

This puzzling observation has not eluded the attention of previous investigators. Conner[8] writes, "It is not possible here to enter into a detailed discussion of the causation of such bronchiectases. The surprising thing is not that they occasionally occurred, but that they should have been found so infrequently. In all but a few of the cases of tracheobronchial syphilis (perhaps 15 per cent), obstruction of some sort existed, and yet dilatation was found in only 15 per cent of the autopsies. That is to say, in not more than 20 per cent of the cases of permanent stenosis did bronchiectasis occur. It seems evident, therefore, that the tracheal or bronchial stenosis is not the chief determining cause of the bronchiectasis found. Much more potent factors, probably, are the changes occurring in the bronchial wall and in the adjacent lung tissue by which the muscular and elastic tissues of

the bronchi, upon which their strength and resilience depend, is replaced by inflammatory tissue." He then cites autopsy records of two cases of tracheal stenosis in which dilatation was confined to the bronchi subserving a pneumonic lobe while other bronchi escaped; one case of stricture of a left main bronchus in which dilatation was confined to the branches to a sclerotic upper lobe and one of general bronchial dilatation in which no stenosis was found and no obstructive symptoms had been present.

Inflammatory processes, softening the alveoli and bronchi and robbing their walls of their normal elasticity probably play a considerable rôle; probably also the varying rigidity of various parts of the respiratory tract. Infections underlying these inflammatory processes would cause a purulent secretion from the bronchial wall, leading to dilatation if drainage is obstructed; aseptic blocked areas would contain no pus or secretion of any kind. After the air in them is absorbed they would collapse and remain empty. There may also be a certain vicariation between bronchiectasia and emphysema. It may well be, as Orth asserts, that with a resilient, air-containing parenchyma the respiratory tractile forces will dilate the softer alveoli, producing in the end an emphysema, but if the alveolar parenchyma has lost its expansibility either through atelectatic obliteration or through chronic suppuration and sclerosis, then the chest wall dilates the bronchi, finding no intervening expansile structure upon which to act.

Thus if the stenotic lobe is hard and airless the bronchi are dilated; if it is soft and air-containing, not the bronchi but the alveoli.

Exactly as in other organs so in the lung, peripheral dilatation may accompany a partial block but not a total one; total block causes not dilatation but shrinkage; air is absorbed from that portion of the lung to which no further air has access; the lung collapses and shrivels. In this totally atelectatic area the bronchi are often found dilated, perhaps by the force of pent-up secretions, perhaps by the traction of the thoracic wall and diaphragm.

The foregoing ideal theoretical postulates are rarely encountered in vivo. Septic processes, if they do not cause the underlying stenosis, rarely fail to complicate it, and sooner or later instead of an aseptic air field blocked by a constriction we have purulent bronchial secretions with the formation of abscesses, ulceration and pneumonic infiltration. The pathologic anatomy and physiology of constriction of the air passages are therefore involved, not only because of the complexity of respiratory aerodynamics but because the restricted respiratory movements are soon complicated by further factors impossible to calculate.

Between total bronchial obstruction with atelectasis and partial obstruction with dilatation stands chronic intermittent obstruction which

gives rise to a most striking and characteristic clinical picture. Some intermission in degree of obstruction is encountered in the course of many stenoses, even in stenosing tumors and other processes that end in total block. There comes a time in the course of a progressive stenosis when the step from partial to total obstruction is very short, and when slight complicating factors, a little more or less swelling of the bronchial mucosa, a little more or less viscid secretion, a little more or less congestion of a tumor will alternately completely block the bronchi and leave a certain portion of their lumen free; the lung then passes from a state of dilatation to atelectasis; correspondingly one finds it on one day filled with air, and the next with fluid; huge amounts of secretion may be brought forth at one period and none the next. With the change in lung volume the mediastinum makes extraordinary shifts. This will be discussed more fully later.

MORBID ANATOMY

Tracheal and tracheobronchial obstruction involving the air supply to both lungs presents varied anatomical pictures. In syphilis, a common cause of obstruction, the tracheal mucosa may be eroded by larger and smaller ulcers, some more superficial, others deeper, destroying the cartilages, which may lie loose in the tracheal wall. The ulcers may be covered with a dingy fibrinous deposit, or form the entrance to a deep caseous cavity. These gummatous infiltrates may perforate the mediastinum and greater vessels. Some ulcers show a tendency to heal and have scarred edges; others heal entirely, so that not the ulcer but a tough scar stretches across the trachea and constricts its lumen as a ring, a band or a spiral thickening of the wall. Huge dense peritracheal and peribronchial syphilitic deposits are not infrequently described. To judge from reports in the older literature, syphilitic constrictions are no longer so common as they used to be. Vierling[55] in 1858 could collect 46 cases, and Conner[8] in 1903, 82 additional ones; among well over a hundred cases of stenosis I have seen but four that were attributable with certainty to syphilis.

Primary Tracheal Tuberculosis is very rare. v. Hansemann[56] reports one and Schmorl[57] two cases. The larynx usually participates in tuberculosis of the trachea and bronchi. Tuberculosis of the trachea and bronchi without laryngeal involvement occurs in about 4 per cent of all cases of pulmonary tuberculosis. Diffuse tubercular deposits in the tracheal and bronchial walls are often 1 cm. thick and greatly obstruct the lumen. In a case of my own the trachea was much thickened; the wall was the seat of a submucous deposit so thick that Dr. Ophüls on first looking at it thought of a malignancy. While primary tubercular infiltrates without ulceration and perforation are rare, ulceration from tubercular peritracheal lymph glands and tubercular peritracheitis are among the most common causes of obstruction.

The tracheal ulcer may lead to a cheesy glandular abscess discharging its contents into the tracheal lumen, the discharge not uncommonly containing chalky débris (lung stones), a periadenitic infiltrate may gradually erode the tracheal wall and show itself as a knob of granulation tissue sprouting into the lumen, and later a whole gland may be delivered into the trachea through this opening. Like syphilis, tuberculosis may make a dense peritracheal or peribronchial infiltrate without suppuration. Both tuberculosis and syphilis may involve the trachea, the tracheal bifurcation and extend along one or both major bronchi, but very extensive infiltrates, especially those at the bifurcation, are more likely to be syphilitic than tubercular.

Rhinoscleroma and leprosy make hard infiltrates and multiple scars. Both usually go hand in hand with laryngeal changes but may affect the lower air passages alone. The tumors of rhinoscleroma are described as diffuse, columnar often symmetrical granulomas, nodular, rarely polypoid, occurring mainly on the posterior tracheal wall. They are greyish red and soft at first, later harder and more lardaceous, and finally very hard. They lead to funnel-shaped and annular stenosis, often excentric, often multiple, characterized by constant absence of ulceration. *Glanders* may attack both larynx and trachea. It makes ragged undermined ulcers, erodes the cartilage and causes perichondritic abscesses. The ulcers are indistinguishable clinically from tubercular ones. They are usually fatal; they may heal with constricting scars.

Typhoid, actinomycosis, and other rarer suppurations have already been mentioned. Actinomycosis does not attack the mucosa primarily, but it may make excessively hard peritracheal infiltrates (woody phlegmons) perforating into the lumen with multiple fistulas. Actinomycotic granulations bleed readily and profusely.

The anatomy of an interesting series of tracheobronchial deformities has been described by Schmorl[27] and Gey[58] under the name of "Bronchitis Deformans." This chronic inflammatory process affects adults mostly males, over fifty-five, frequently persons who follow dusty occupations. It involves mainly the upper and middle bronchi, occasionally also the trachea, and the right lung more often than the left. It gives rise to dense peribronchial deposits which kink and distort the bronchi and constrict their lumen. Ulceration is infrequent, the bronchial wall need not be much hypertrophied, but it is dilated in places, shrunken in others, angulated and indented by the dense scar that surrounds it. The mucosa is thick, nodular and uneven. The submucosa is frequently stained by deposits of anthracotic pigment carried over from adjacent lymph nodes. The peribronchial glands are usually sclerosed, hard, and firmly attached to the bronchial wall. They occasionally suppurate and perforate into the esophagus, medi-

astinum and great vessels, and induce communication between the trachea and these structures. The changes found at autopsy are probably the result of chronic intermittent processes, fresh ulcerating foci alternating with old scars.

Schmorl found small fresh tubercular ulcerations distal to the stenosis. He considers this stagnant area favorable to the implantation and development not only of tuberculosis but also of cancer. He included no less than five cancers in his series. Perforation into the bronchial lumen is always the result of acute suppuration; staphylococci and streptococci, but not tubercle bacilli, are found in the exudates. However, if the perforating gland is the seat of old tubercular foci, tubercular pneumonia may occur from aspiration of its contents into the smaller bronchi.

The symptomatology of this picture is exactly what one might expect from Schmorl's classical description.

CASE 16.—On Sept. 19, 1926, Dr. Stewart Irwin of Oakland asked me to see a Hollander of fifty-eight who had worked as a mining engineer, but never underground. Fifteen years previously he had been brought down from the mines with what was probably a thrombosis of the cavernous sinus complicating a mastoiditis. He was operated upon and recovered after six months of precarious illness.

For from three to five years he had had a cough with profuse, but never very foul expectoration and much dyspnea. He had had several attacks of high fever (to 103° F.), the last one confining him to bed from November 25 to December 24, 1925. Fever, increase in cough and expectoration came on suddenly in three or four days. He had no pain. When Dr. Irwin first saw him three years ago the lung fields were clear, except for some peribronchial thickening, but the mediastinal glands were so enlarged that Dr. Irwin thought the man had Hodgkin's disease. Many examinations of the sputum failed to demonstrate tubercle bacilli.

I found a man of robust bones but poor muscles, with a yellowish skin, who was dyspneic even when lying quietly on his back. He coughed when he moved and brought up much yellowish sputum. The right chest moved less than the left; there was a dullness around the right hilum; the right base was flat. The intercostal spaces were not retracted or tender. The breath sounds were diminished over the right base; over the right hilum and in patches over the base they were amphoric. There were moist râles at both bases, more on the right than on the left. The liver projected an inch under the ribs.

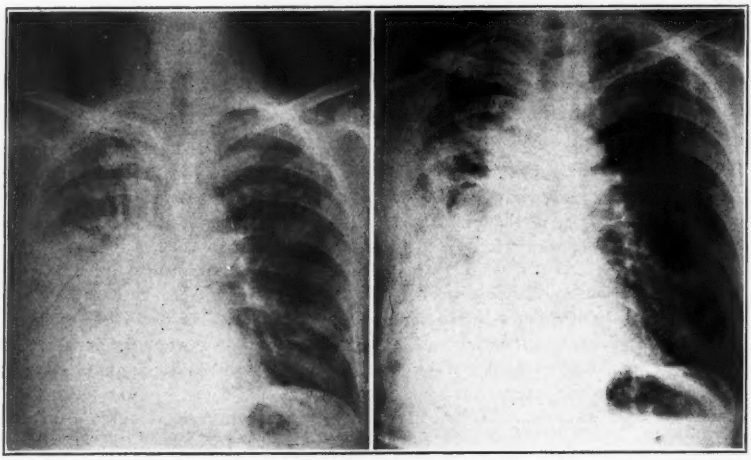
Films taken in September, 1926, showed an impenetrable greyness at the right base, with discrete clear areas, possibly cavities, above. The heart was flat; above it, but too low for the aortic arch, lay a round shadow with a border unusually smooth and regular for a glandular opacity.

The patient was taken to Stanford Hospital where lipiodol injected through the cricoid membrane demonstrated a normal left lower lobe, but some cylindrical dilatation of the right lower bronchi. Very little lipiodol entered the right middle lobe. In films of October 14, the mediastinum was almost in a normal position, but in films of October 12 and October 15 it was displaced markedly to the right. The right diaphragm was high and moved very little; there was a suggestion of paradoxical movement. The evidence of intermittent stenosis strongly suggested a neoplasm at the right lung root, most likely a bronchial carcinoma.

The sputum contained streptococci, a few fusiform bacilli and spirochetes; no tubercle bacilli were found.

Bronchoscopy by Dr. Sewell on October 16 revealed an apparently normal left main bronchus. The right main bronchus was swollen in its anteromesial aspect; its mucosa bled when touched. The anteromesial aspect was encroached upon as though by a growth, and it was irregular. Mucopus was aspirated from the right lung.

On October 23 about four inches of the sixth, seventh and eighth ribs were resected posteriorly. The pleura was ⅛ inch thick; the lung was adherent and pulled tightly on it. A needle encountered an induration but withdrew no pus. A large flap of pleura was opened, whereupon a central induration the size of a golf ball could be felt through the peripheral soft lung. A funnel-shaped entrance was made with a galvanocautery and an abscess containing green but not foul pus was opened. The wound was packed.



Right Fig. 27. Left Right Fig. 28. Left

Fig. 27.—Case 16. Sept. 8, 1926: Large round shadow lies in left hilum above heart. Right chest contracted; right base is gray; heart and mediastinum drawn to right.

Fig. 28.—Case 16. Oct. 15, 1926: Right chest is clearer; heart and mediastinum are still much retracted to the right.

A few hours later the patient developed epileptic seizures; the temperature rose to 109.4° and he died the next morning of cerebral embolism.

Autopsy revealed aortic emboli and an embolus of the left internal carotid artery.

The left pleura contained no fluid; the left lung was large and emphysematous anteriorly and in the upper lobe.

The right pleural cavity was obliterated by dense adhesions, several millimeters thick. The cut surface of the right lung was firm, with scattered areas of consolidation and fibrosis. The lobes were strongly bound together. At the hilum a branch of the first order of the lower lobe bronchus was constricted for a distance of 1 to 2 centimeters, its lumen being narrowed to about one-half of normal. At this site the mucosa was rough and congested and the wall was bordered by a number of enlarged lymph nodes containing greyish areas. The cartilages of the narrowed portions were calcified and much thickened. The stenotic bronchus led to a ragged, clean, funnel-shaped cavity (the former abscess) measuring 5 cm. by 5 cm., with a base at the periphery of the lung.

Microscopically the lungs revealed fibrosis and scarring, but no tuberculosis; the peribronchial lymph nodes also were fibrotic, but not tubercular.

Paraffine sections of the bronchus at its narrowed part showed marked fibrous thickening of the deeper tissues with round cell infiltration. The mucosa was greatly thickened and congested. One half of a cartilaginous ring in cross-section was composed of hyalin cartilage, the other half of a thin shell of bone surrounding well-developed bone marrow. The two ends of the semicircle of bone joined the cartilage abruptly.

The anatomical diagnosis read: Abscess of lung, due to stenosis and plugging of a large bronchus; embolism of left internal carotid artery.

Perforations of the bronchi into the esophagus are evidently the result of processes similar to those described by Zenker[59] and others as precursors of traction diverticula.

Intrinsic stenosing tumors, aberrent thyroids and tracheal chondropathies have been described above.

Lungs subserved by an obstructed trachea or by a constricted bifurcation are often museums of pathology. They may contain tubercular cavities, fibrous syphilitic pneumonias and other evidences of disease related to the constricting process. Aside from this the pleura is usually more or less thickened and adherent. Often pneumonic areas are found, varying in size, degree of consolidation and age, which not uncommonly represent the immediate cause of death. These may surround foul liquefied gangrenous vomicas or abscess cavities. They may be studded with minute miliary abscesses. Areas of bronchiectasis are rarely wanting, but as has been mentioned before, their distribution is unequal; fairly normal lobes, or portions of a lobe vary with others almost devoid of spongy parenchyma and occupied by large bronchiectatic cavities, sometimes round or saccular, more often tubular or bottle-shaped. At times merely thin fibrous sheets separate one large cavity from another; more often clubbed bronchi end in a thick, leathery, grayish, airless field of fibrous pneumonic infiltration. The walls of such bronchi are usually thick and stiff; they thread their way to the hilum as stiff white tubes. Their lining is thickened; they are filled with viscid purulent mucus. Often one lung is notably more affected than the other; the mediastinum is then drawn toward the more affected side, the lung shrunken and covered with a thick pleura; in such a lung bronchiectasis is likely to be saccular and diffuse. At times the dilatations are so extensive that the whole lung is converted, not into a honeycomb, but rather into a shell filled with great bronchiectatic bubbles. It is not unlikely that many such lungs are the seat of congenital malformations, and that the stenosis is contemporaneous with the bronchial dilatation and a result of the same embryonic malformation rather than a cause of the dilatation, or that a primary process causing atelectasis, such as embolism, has led to fibrosis and secondarily to both bronchiectasis and stenosis, each abetting the other.

In both bilateral and unilateral stenoses the hilum glands are regularly enlarged, sometimes soft, sometimes hard. The pleura is likely to be thickened and adherent, but enormous pleural deposits with obliterations of the whole cavity occur not in bilateral, but unilateral and unilobar stenosis.

The heart is often dilated, especially the right side; the liver and spleen are congested, but edema and visceral collections of fluid are not frequent.

The effects of *partial unilateral stenosis* involving one of the major bronchi, uncomplicated by infectious elements may best be studied in infantile congenital stenosis. The lung is enormously dilated, driven so far to the other side that it has been mistaken for a spontaneous valve pneumothorax. Later in life, as fibrous pleurisy supervenes, the lung shrinks. It may be divided into two or three large unilocular cysts, each corresponding to a lobe, or into large multilocular cystic sacculations. A number of such cases have been described in infants. (See Case 1.)

In *acquired unilateral stenosis* affecting one of the major bronchi the original anatomy is obscured by superimposed complications. The bronchus may be occluded by a tumor, either metastatic, involving the hilum glands and compressing the lumen, or originating in the intrapulmonary part of one of the large bronchi and gradually invading the major ones. In such cases the bronchus may lie embedded in hard tumor masses, not infrequently ulcerating into the lumen and obstructing it with a foul, suppurating, crumbly growth. Not infrequently compression is due to a cancerous peribronchial infiltrate. Pulmonary cancer seems particularly prone to invade the peribronchial lymphatics and to encroach via this route onto the hilum, the great vessels and the heart itself.

The lung of an inflammatory stenosis is not dilated as in congenital stenosis but much shrunken. The same inflammatory processes that cause obstruction excite pleurisy, fibrous and shrinking pneumonia. The mediastinum is drawn to the affected side, the pleura thickened, and as in the lungs described before, patchy red areas of acute pneumonia, abscesses, gangrene, gray, airless leathery areas of chronic fibrosis, and tubular and saccular bronchiectasis variegate the cut section. Occasionally bronchial dilatation may be extreme and almost universal, and the lung may be converted into a conglomeration of huge bronchiectatic sacs filled with thick, slimy pus.

CASE 17.—A fireman of forty-four was referred by Dr. G. D. Schoonmaker on February 24, 1927. One night a year before he had come home complaining of a chill. About a month later he began to cough up a sputum which was at first slimy, later purulent, and frequently blood stained, but never very foul. The man had lost forty-five pounds. Since December, 1926, he had been hoarse and aphonic. At examination he was thin and pale. The left chest lagged and was

dull from the scapula downward. The breathing was amphoric over the sternum; loud and amphoric over the lateral part of the left chest. The lower left intercostal spaces were tender to pressure.

Lipiodol injection showed marked deviation of the trachea to the left and a stenosis of the left major bronchus about 1 to 1½ inch below the bifurcation. Bronchoscopy by Dr. Fletcher revealed:

"Recurrent laryngeal paralysis, left side. Small, healed granulation tab, left side of larynx above vocal cord. Tracheal rings normal, but position of trachea above bifurcation is curved to left instead of being straight. The carina is somewhat less sharp than normal. The right main bronchus appears normal. The left main bronchus appears more or less edematous throughout with very few

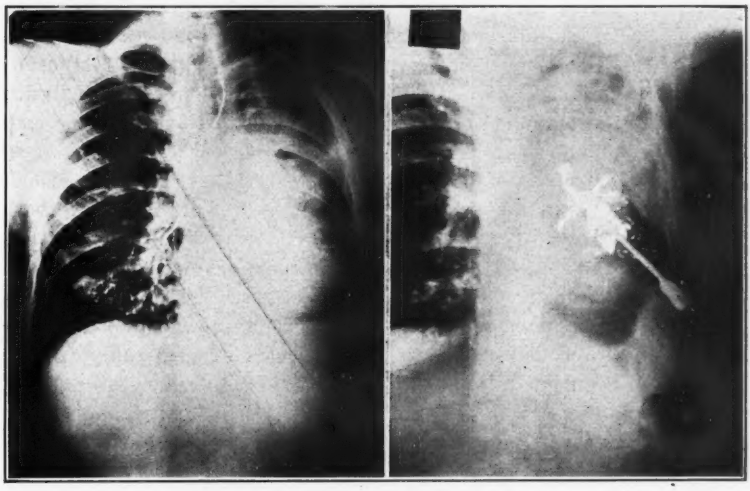


Fig. 29. Fig. 30.

Fig. 29.—Case 17. Bronchial stenosis, left, before operation, showing marked tracheal deviation. A small spot of lipiodol may be seen filling the origin of the left major bronchus; the rest of it has spilled over into the right base.
Fig. 30.—Case 17. Same after operation. Stenosis filled from periphery.

cartilaginous ring markings which disappear entirely after entering about 2 cm. The left main bronchus then ends about 4 to 5 cm. beyond the bifurcation in a tube completely closed by what appears to be edematous tissue. A soft bougie was inserted into the closed lumen about 1½ inch, no definite obstruction being apparent either on insertion or removal. There was slight bleeding after withdrawal of the bougie.

"The impression gained was that the lumen was closed by edematous membrane with probably some pressure from without.

"The distance from the bifurcation to the end of the bronchoscope at the lowest point it could be inserted was between 4 and 5 centimeters."

On March 11, 1927, the seventh, eighth and ninth ribs were resected. The pleura was adherent. The lung was entered with a Percy cautery. Much grumous pus exuded from the cauterized lung. Pus continued to discharge through this opening, but only a little air came from it after cough. An attempt at bronchoscopy was unsuccessful. A bronchoscope passed through the fistula revealed a hard, pulsating mass in one of the bronchi of the second order. Glandular metastases

developed in the left axilla and the man died July 7, 1927. The final diagnosis was carcinoma of a left bronchus with stenosis at or near the bifurcation. Autopsy was not permitted.

A single lobe depending from a totally stenotic bronchus when seen at operation or autopsy is dark, plum-colored, congested, its pleura usually adherent. The adhesions are usually very vascular; enough so to require ligation if cut. Those to the parietal pleura are dense and separated with difficulty; if there is underlying suppuration the lobe is often torn in an attempt to separate them. Interlobar adhesions are usually less firm; they are vascular mainly at their edges; toward the hilum they are ordinarily easily separable. This is also true of pericardial adhesions, fortunately, for there is little danger of entering the heart sac when separating them at operation. Diaphragmatic adhesions, on the other hand, are peculiarly dense; they will not cleave, and must be cut; other than careful dissection will penetrate the diaphragmatic muscle fibers, which are often brown, thin and atrophic. The totally stenotic lobe itself is leathery, much shriveled, perhaps $\frac{1}{5}$ to $\frac{1}{10}$ its normal size, and airless. It is often so small that it is hard to recognize. It may, and usually does, if an infection has complicated the stenosis, contain larger or smaller collections of pus, either in bronchial pockets or as true abscesses. The bronchi themselves are usually somewhat thickened, and if a period of partial obstruction has preceded the total one, they may be considerably dilated. If the main bronchus is free and only a part of its branches are obliterated, part of the lobe may be atelectatic and the remainder soft, pink and air-containing. This is clearly demonstrated at operation and occasionally by the x-ray. The adhesions over the air-containing part are thin and frail; those over the collapsed part dense and vascular.

CASE 18.—A man of twenty-five was admitted to the San Francisco Hospital in July, 1927. He had had pneumonia in 1917 and influenza in 1920. In 1923 he brought up some three ounces of blood and bled about once a month up to 1924. Since then he had coughed but had no further hemorrhage. On admission he coughed incessantly; the right side of the chest lagged; the right apex was dull and the breath sounds tubular. On July 15, 1927, Dr. Graham did a bronchoscopy and found a polyp attached to the lateral wall of the right major bronchus, which he removed. The man improved and continued to work as an orderly in the hospital, reentering at intervals on account of cough and fever. Several bronchoscopies were done, finally on January 7, 1929, a bronchoscopy was done and a bit of tissue removed, which was diagnosed as carcinoma. A densely adherent carcinomatous upper lobe, the medial part of which was airless and infiltrated, was removed, but the man died of shock. Autopsy was refused.

An incompletely obstructed lobe presents much the appearance of the lung described above under unilateral stenosis. The pleura is thick, adherent, and vascular; the size of the lobe is usually not increased but diminished by the scars of preceding infections. Its surface is of

varying consistency, hard areas alternating with soft ones that betray underlying cavities. The constricted bronchus is as a rule easily demonstrable in the fresh specimen, and so is the cause of the constriction—glandular masses, a tumor, etc. In more recent obstruction peripheral bronchial dilatation may be less marked. The bronchi are thickened, larger and smaller abscesses are scattered through the periphery of a gray, partly suppurating lobe; purplish pneumonic patches may surround some of the pus-containing bronchi. Older obstructions are likely to have dilated the peripheral bronchi into bottle- or club-shaped receptacles, each containing a quantity of mucopurulent secretion. The bronchial walls are gray and glistening or red and inflamed, thick and encased in a firm, fibrous coat. Between

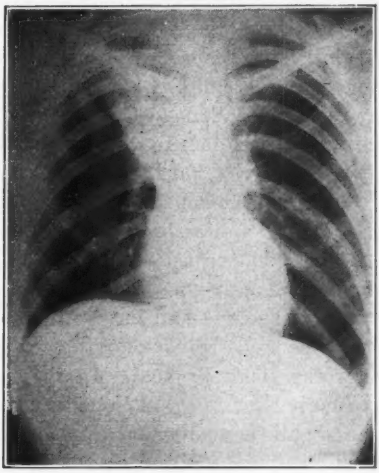


Fig. 31.—Case 18. Stenosis of part of right upper lobe. Bronchial carcinoma.

them lies dense, gray, firm fibrous lung; or (if the dilatation is excessive and saccular) a partition-work traversed by larger blood vessels and small bronchi, with strands of black anthracotic pigment streaking the white section of the outer layers of the bronchial wall. The bronchiectatic cavities stand out most strikingly if the trachea is injected postmortem with a hot barium-gelatin mixture.

Near the hilum the large bronchi are thick walled, stiff and white; between them lie large, soft black lymph glands and sectors of gray, more or less airless, or reddish-purple and more or less pneumonic lung.

Between the two extremes described above lie all manner of variations, degrees, and combinations. Older and more extreme, and recent and less marked dilatation, inflammation and suppuration may lie side by side in the same lobe.

In intermittent stenosis of the larger bronchi the picture changes accordingly as the stenosis is open or closed. Behind a closed stenosis the lobe may be more or less acutely pneumonic, red, carnefied from preceding attacks, the bronchi filled with thick pus; when the obstruction is open the lobe contains air and is less shrunken, but the bronchi are still dilated, inflamed, and contain secretion.

Stenosis of the smaller bronchi is diffuse, affecting both sides of the chest in the cases that have come under my own observation; whether it occurs unilaterally and whether the dense unilateral shadows that are sometimes seen in films of pulmonary tuberculosis may not be due to this cause, I do not know. Diffuse chronic stenosis is intermittent in severity and of necessity incomplete.

Sergent[32] and Benda[31] have described a chronic syphilitic bronchitis, agreeing in some details with the picture described under Case 13. They state that syphilis may cause a dense peribronchial sclerosis which, in distinction to the irregularly disseminated lesions of tubercular fibrosis, characteristically follows the topographical distribution of the bronchial tree from the hilum to its finest branches. The syphilitic deposits are extraordinarily hard, sometimes encase the bronchi, sometimes vegetate into their lumen. They may leave the lumen unchanged, they may dilate it, and again, they may constrict and obliterate it. Scattered about in the sclerotic deposits are more or less numerous older and fresher gummatous nodules. The lungs are extremely hard; both their surface and cut sections are studded with granules the size of grape seeds which represent ends and cut sections of indurations about the small bronchi. In the lower lobes Benda found small emphysematous cysts. Not infrequently tuberculosis and syphilis coexist; the syphilitic component imprinting its sclerogenous qualities onto the tubercular one gives the tuberculosis a characteristically chalky and fibrous form.

Stenosis of the bronchioli (Bronchiolitis fibrinosa of Fraenkel[9]) is also a diffuse process, more or less patchy. It may occur after inhalation of destructive gases or infections. It has been described after influenza. The lungs are much dilated and do not collapse after the chest is opened. Under the pleura there lie innumerable minute dark red spots, exactly like miliary tubercles. Examination with a magnifying glass, however, shows them to differ from tubercles in being not round but stellate. The obstructed bronchioli appear in the cut sections as fine gray branching streaks, 0.5 mm. wide. The fact that no miliary tubercles are found in other organs will at once excite suspicion that the disease is not tuberculous.

SYMPTOMS

Among the *symptoms* of bronchial stenosis *dyspnea* plays a prominent but varying part. In *high stenosis of the trachea or both bronchi*

at the bifurcation breathlessness is very apparent on exertion, although a patient lying quietly in bed may seem to breathe quite freely. Gerhardt[7] divides stenotic symptoms into three periods: the irritative, the period of constant dyspnea, and the period of suffocative attacks. The first two stages are often, but not always recognizable; cough often, but not always, precedes dyspnea.

During the stage of constant dyspnea inspiration is prolonged, difficult and wheezing; expiration is usually little changed. At times dyspnea is apparent only on exertion, at times it is constant. Often the patient complains but little, while at the same time his breathlessness may be so apparent and distressing to look upon that it is hard to understand how a man can struggle so for breath and still live.

Observations of the time of expiration and inspiration in stenosis are few; Connor[8] in his 82 cases has none. In accuracy modern observers must yield to Mr. Worthington[60] who in 1842 writes:

"Charles Newrick, aged forty-nine, an agricultural laborer, of spare habit of body, first came under my notice in August, 1837. During the early part of his life, he had enjoyed tolerably good health, with the exception of occasionally suffering from slight cough, which was sometimes increased by the employment of threshing, owing to the dust rising from the corn, and irritating the fauces and respiratory passages. In 1833 he contracted syphilis, for the cure of which mercury had been administered, but not to an immoderate extent. At this time he experienced an increase of cough, and soreness about the throat, attended by slight difficulty of swallowing. His general health also began to decline, as denoted by occasional feverishness, impaired appetite and loss of flesh. These symptoms progressively and steadily advanced until the time of my first seeing him (August, 1837), when I found him suffering from the following symptoms: During the last twelve months he had been confined to the house; he was much emaciated, was very feeble, and complained of want of appetite, and of uneasiness about the throat. His state of breathing more particularly arrested my attention, as regarded both the peculiarity of the noise attendant upon inspiration, and the very painful effort required for its accomplishment. In the ordinary act of inspiring, a sound was produced, exactly resembling that produced by an unsound horse, called a roarer or whistler, and this sound at once suggested the idea that, in the act of inspiring, the air passed through a tube of preternaturally small diameter. Each inspiration occupied ten seconds, the chest expanding only six times in a minute. Expiration was performed in much less time than inspiration, with much less exertion and with diminished intensity of roaring. Upon examining the muscles of the throat and neck, I was forcibly struck with their violent action, more particularly with that of the sternohyoidei and sternothyroidei muscles, together with the thyro- and omohyoidei and other muscles of the larynx, whilst the trapezeii intercostales and diaphragm were comparatively much less acted upon. From this circumstance, aided by other symptoms, I could not fail of concluding that the impediment to the free passage of air into the lungs existed within the trachea or larynx, but I could not satisfactorily determine as to which of the two was principally affected. Vocalization was very imperfect, the sound of utterance being hoarse and rough. A troublesome cough was present, accompanied with a copious mucopurulent expectoration, the checking of which tended, in some degree, to increase the difficulty of breathing. The patient complained also of an offensive discharge from the nostrils, followed by occasional exfoliation of osseous matter,

which appeared to be connected with disease of the inferior turbinated bones. The pulse was small, quick and irritable, but no well-defined symptoms of hectic fever had yet supervened. The larynx, when moderately compressed, did not evince pain and it was only when an increased force was used that uneasiness was occasioned. The mouth and fauces did not present any marks of disease, either of the tonsils, uvula or palate, nor did these parts appear to have been the seat of previous ulceration. A slight roughness of the epiglottis was detected by the finger, but the stethoscope applied over the chest furnished no indication of disease of the lungs.

"I was convinced that little or nothing beyond a palliative treatment was applicable to this man, and I ordered him a light nutritious diet. I prescribed some sedative and expectorant medicine, placed him as an out-patient of the infirmary, and occasionally visited him. Toward the autumn of the same year (1837), he so far regained strength as to walk into the air and to present himself at the infirmary, and he continued to do so occasionally for nearly four years, in the course of which time his local symptoms did not vary much. The peculiar roaring sound never left him, nor the abnormal state of his voice, both symptoms maintaining the same character as when I first saw him. He was generally worse when the atmosphere was damp and cold, and when exposed to the night air. In the winter months he was mostly confined to the house, but as the weather became warmer, he could, if allowed to take his own time, walk about three or four miles in the day. Whatever promoted expectoration, usually produced a temporary relief of the dyspnea. He described the expectorated matter as having sometimes assumed an arborescent appearance. His death took place on the 15th March, 1841. The whole of the week preceding the event he had been as well as usual. On the morning of the day of his death, whilst taking some bread and milk for breakfast, some particles of this food got into the larynx, and he was suffocated in less than five minutes.

"*Autopsy twenty hours after death:* The muscles of the anterior aspect of the neck were found unusually developed, their fibers being of a deep red color, and offering a degree of rigidity when cut into, not commonly met with in that situation, and in a subject so slender and emaciated as he was. This development of these muscles manifestly proceeded from the increased action they had performed for a considerable period before death, in overcoming the impediment to the passage of air through the trachea. The lungs were moderately distended, were crepitous, and free from emphysema. The bronchial tubes were filled with viscid mucus, but in no respect morbidly dilated. The glands situated about their roots were enlarged, and one of them particularly, just at the bifurcation of the trachea, was found to contain calcareous deposit, but did not appear to have produced any undue pressure upon the parts around it. The heart was smaller than is usually met with in adults, and the pericardium contained about two ounces of serum.

"The trachea was detached just below its division, and removed, together with the larynx, for the purpose of close inspection. Having deprived them of their surrounding tissue, a singularly well-defined constriction, constituting complete stricture, was discovered just below the cricoid cartilage, the calibre of the strictured portion not exceeding that of a crow-quill, and at once disclosing the principal cause of the distressing symptoms during life. This partial obliteration of the canal was independent of any adventitious membrane, the product of either acute or chronic inflammatory action, as in croupy affections, and of the existence of any of the usual marks of inflammation. The tracheal rings, at the point of stricture, had entirely disappeared, and had been converted into a fibrocellular tissue, whilst those below the constriction were much dilated beyond their natural circumference, and had also to a certain extent lost their elastic and cartilaginous

character. The larynx, when held perpendicularly, presented a more flattened appearance than natural, owing to the approximation of the alae of the thyroid cartilage. This altered shape may probably be regarded as a consequence of the stricture in the trachea, and it no doubt in some degree added to the difficulty of breathing. The epiglottis showed marks of having been attacked with ulceration at some former period; the only vestiges of it remaining were two or three small irregular vegetations. The lining membrane within the larynx was slightly thickened, pale, and rather thickly smeared with a viscid muco-puriform fluid, but it presented no appearance of ever having been the seat of ulceration.

It is extremely probable that the tracheal disease had a syphilitic origin; and that the absorption of the cartilaginous rings of the trachea in the seat of stricture had caused the constriction, the contractility of the circular or transverse fibers of the membranous portion of the trachea having been no longer antagonized by these cartilaginous rings. This seems an obvious solution of the production of the organic lesion displayed in this case, which illustrates several points in the mechanism and physiology of the respiratory passages. I have never met with, nor read of, a similar case to the present: and my friend Dr. Copland, when lately in this part of the country, informed me that he was not acquainted with any instance on record, in which the same changes as the above had been observed upon dissection."

NOTE BY DR. COPLAND

"By permission of Mr. Worthington, the trachea, sent by him with his paper to the Society, was slit open by Mr. Shaw. Its inner surface presented superficial cicatrices extending both below and above the strictured portion. The cicatrized surface was smooth, although somewhat irregular, and of a serous or polished appearance; showing that the ulceration had healed long previously to death. The cartilaginous rings of the trachea were entirely absorbed from about half an inch below the thyroid cartilage downward to the extent of about three inches. The upper part of the trachea, that had thus lost the antagonizing power to the transverse fibrous structure, was constricted so as to admit only a crow-quill. The inner surface of the constricted part was quite smooth. The larynx was sound; but the inner surface of its base and the commencement of the trachea presented superficial, slight and old cicatrices. The trachea was much dilated from a little below the strictured part, to the bifurcation, and its internal surface presented the superficial cicatrices already mentioned."

Infection, especially pneumonia increases the respiratory rate. What part of the increase is due to blocking of the lung, what to infection and toxemia, and what to cardiac insufficiency, is difficult to say.

A number of observers have busied themselves with respiratory studies in emphysema, but accurate spirometric observations of stenosis as it occurs in nature are wanting. Forschbach and Bittorf[48] let normal individuals breathe through small openings. They distinguish two types of stenotic respiration. In the most frequent the respiratory rate is unchanged but the mid-position between inspiration and expiration is elevated, owing to a decrease in expiration. In the second type the mid-position is raised through increase of inspiration, but the amount of air exhaled is equal to the amount inhaled. This type seems the more usual in chronic stenosis as seen at the bedside.

It is registered in Fig. 25, page 34, a tracing of Case 7 (tracheal stenosis through pressure of tubercular peritracheal lymph nodes). Numerous animal experiments have shown that inspiration is prolonged and intrapleural pressure lowered in high stenosis. These results, however, may not apply to chronic tracheal stenosis in man.

The third stage of Gerhardt is usually clearly defined. The suffocative attacks are so appalling that no observer, much less the patient, could ever forget one. A constant moderate breathlessness is interrupted by the most alarming attacks, provoked by exertion, or often by no apparent cause at all. In these respiration is gasping, shallow and extremely rapid; the face is gray; the patient breaks out in a

Fig. 32.—A suffocative attack.

Fig. 33.—A suffocative attack.

cold sweat; he looks as though death impended, as indeed it often does. Under morphine the attacks may gradually recede. The patient usually sits up in bed with the head stretched forward, as though stretching the trachea eased his breathing.

Accurate observations of the relative time and difficulty of inspiration and expiration in the suffocative stage show that expiratory difficulty exists. Further clinical observation is necessary to determine whether the transition from the second to the third stage is due to change in the character of respiration, and whether difficult inspiration characterizes the second, but difficulty both of inspiration and expiration the third. Whether transition from constant to paroxysmal dyspnea is due to intrapulmonary causes; whether the change denotes that not only inspiration but expiration has become difficult and that the lung has become dilated with air entrapped distal to con-

striction; whether it is due less to changes in the lung than to the heart, and denotes a failing right ventricle, or whether pulmonary and cardiac changes are both and perhaps equally at fault, is difficult to say. However the autopsy of Case 13 seems to indicate that expiratory dyspnea and increased intrapulmonary pressure may have to do with the initiation of the attack if not with its ending. If Thornton and Pratt's[52] statement that air is entrapped distal to constriction is true even of mild constrictions then in severer ones a moment must come when the resiliency of the lung and the chest wall are insufficient to counterbalance the increase in lung volume; when increased intrathoracic pressure compresses the veins and makes entrance of blood

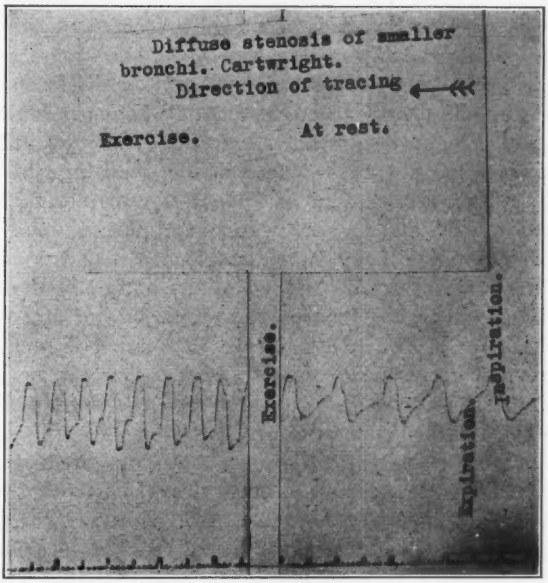


Fig. 34.—Breathing in suffocative attack. Inspiration shortened; expiration relatively increased.

into the chest difficult; when expiratory effort must come to the aid of natural passive forces; there must come a time when these active forces tire and respiratory and cardiac decompensation supervene. I am inclined to think that transition from the second to the third stage is a clinical manifestation of this moment, although I concede that the third stage may be due primarily not to pulmonary but cardiac derangement.

In high stenoses stridor is loud.

When but *one of the major bronchi* is obstructed dyspnea is less severe and is not marked by the stages and attacks that characterize higher stenosis. Patients complain of shortness of breath on exertion

as they do with pneumothorax, large effusions and other lesions that greatly reduce the breathing surface. In one-sided stenosis dyspnea is characteristically increased by attempts at "postural drainage" by laying the patient on the good side with the bad one uppermost. In this position the good lung is compressed and movement of the unaffected side of the thorax is impaired; breathing must of necessity become difficult. In Meyer's[61] case of left-sided congenital cystic lung a suffocative attack could be provoked by putting the infant on its right side.

In *stenosis of a single lobe* dyspnea is not apparent. Patients occasionally complain of feeling as though air failed to reach certain parts of the lung, as though they could not take a deep breath with certain parts of the chest, but even this sign is inconstant.

In *diffuse stenosis of the smaller bronchi or bronchioles* dyspnea is again intense, and is the most prominent symptom. It is constant, interspersed with the most alarming exacerbations, as in high stenosis.

Stridor is loud in tracheal stenosis and often in stenosis at the bifurcation; it is lacking in diffuse stenosis of the small bronchi—a means of differentiation. In unilateral and lobar stenosis although there is no true stridor sticky and clicking, wheezy and purring noises, "music-boxes in the chest" are likely to be disturbing.

Cough is fairly constant. The loud brassy nonproductive cough of mediastinal or tracheal irritation is characteristic of stenosis of the larger air passages. The cough is most distressing in stenosis at or near the hilum or in tracheal stenosis. Such patients wear themselves out coughing. The nearer obstruction of the single bronchi lies to the carina the more likely is cough to be troublesome.

Efforts in mediastinal affections are often nonproductive, sometimes a little thick viscid *sputum*, quite clear, like thick albumen, or purulent, at times bloody, is brought forth. At times the sputum is very copious, watery, frothy, almost like saliva. This probably occurs with obstruction sufficiently marked to cause distal atelectasis and edema. This thin and frothy sputum is often brought up in diffuse obstruction of the smaller bronchi. Hemoptysis is frequent, not due to the stenosis, but rather to the underlying process, a tumor, an ulcerated gland, aneurysm, etc. It may, however, when the congestion is extreme, come from the lung itself. Purulent, sometimes foul sputum is also not strictly a part of the stenotic picture. Expectoration of small chalky particles, "lung-stones" usually denotes ulceration of chalky glands. Repeated sudden variations in the amount of expectoration; sudden floods of thin purulent sputum alternating with a distressing dry cough are characteristic of intermittent stenosis, although this alteration also occurs in communicating suppurations, abscesses, cavities, and empyemas with openings that alternately open and close.

Other complaints are due less to the stenosis as such than to the underlying disease. Pleuritic pain is fairly constant in unilateral and unilobar stenosis, rarer in diffuse obstruction of the smaller bronchi.

Minor symptoms, hoarseness due to tracheitis or to laryngeal palsy, are of value in localizing the site and determining the cause of the obstruction. Swallowing is occasionally disturbed; some patients are able to localize the point at which food sticks. This difficulty occurs not only with esophageal carcinoma and diverticulum but in mediastinal adenopathy and inflammations which interfere with the mobility of the esophagus by causing periesophageal adhesions.

Constitutional disturbances, fever, loss of weight, sweats, fatigue, etc., are not characteristic. High temperature lasting but a few days, accompanied by a marked decrease of expectoration and sometimes, but by no means always, cough occurs in intermittent stenosis, but also in other kinds of incompletely communicating suppuration, such as abscesses or empyema with insufficient drainage.

(*To be continued*)

BRONCHIECTASIS*

(To be published serially in four installments)

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(Continued from p. 193)

II. CLINICAL FEATURES AND DIAGNOSIS

AGE AND SEX

THE age and sex in 182 cases of bronchiectasis which have been observed at the Barnes Hospital Chest Service prior to 1930 have been analyzed. Of these, 149 were considered to be cases of bronchiectasis without gross evidence of any large abscess cavity. They are therefore listed in Chart 1 and Table I as cases of "bronchiectasis without abscess of the lung." It is to be understood that this classification is only for convenience because we hold the opinion that nearly all cases of bronchiectasis are associated with pulmonary abscesses which, however, may sometimes be only microscopic in size. In Table I are listed 33 other cases of bronchiectasis in which one or more pulmonary abscesses were conspicuous features; these are therefore classified as "abscess of the lung plus bronchiectasis." It will be observed that the lesion was as frequent in men as in women. Most cases concerned individuals under forty years of age. We have given the age of the patient at the time of the first observation. The onset of symptoms in many instances antedated these observations by fifteen to twenty years. We do not know whether the reason why so few patients with bronchiectasis are observed after 60 is because they succumbed before that age period or because they merely failed to present themselves for observation because they were content to carry on as they were.

LOCATION

The location of bronchiectasis in 149 cases has been tabulated in Table III. It is to be noted that the lesion was unilateral on 83 occasions. Most of the patients referred to were subjected to lipiodol injections. It can still be said that bronchiectasis is essentially a disease of the lower lobe. Apical bronchiectasis should always arouse the suspicion of tuberculosis. Individuals suffering from chronic fibroid phthisis always have some degree of both apical and basal bronchiectasis. The latter need not always be of a tuberculous nature. Congenital bronchiectasis is frequently unilateral and usually involves the left lower lobe.

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TABLE I

BRONCHIECTASIS—NONTUBERCULOUS WITHOUT ABSCESS OF THE LUNG
(See text for explanation of classification)
Number of Cases 149

MALE	ETIOLOGY	AGE PERIOD	FEMALE	ETIOLOGY
9	1 Pertussis 2 "Measles" (Pneumonia) 1 Scarlet Fever 1 Foreign Body 2 "Colds" 2 Uncertain	1-10	4	1 Pertussis 1 "Measles" 1 Pneumonia 1 Uncertain
13	2 Pertussis 2 "Colds" 5 Influenza 2 Pneumonia 2 Uncertain	10-20	17	7 Pertussis 1 "Colds" 4 Influenza 3 Pneumonia 1 Posttonsillectomy 1 Uncertain
17	7 Influenza 2 Pneumonia 1 Foreign Body 1 "Measles" 2 "Colds" 1 Spirochetosis 1 Chest Injury 1 Tooth Extraction 1 Broncholithiasis	20-30	16	7 Influenza 3 Pneumonia 2 "Colds" 1 "Asthmatic Bronchitis" 1 Syphilis 2 Uncertain
13	2 Influenza 1 "Colds" 2 Pertussis 2 Broncholithiasis 1 "Asthmatic Bronchitis" 1 Congenital (?) 1 Foreign Body 1 Posttonsillectomy 2 Uncertain	30-40	15	2 Influenza 4 "Colds" 1 Pertussis 1 Broncholithiasis 1 "Asthmatic Bronchitis" 2 "Chronic Bronchitis" 2 Uncertain 2 Pneumonia
7	2 Influenza 1 "Colds" 1 Syphilis 1 "Asthmatic Bronchitis" 1 postcholecystectomy 1 Uncertain	40-50	10	1 Influenza 2 "Colds" 1 Syphilis 1 Anthracosis 2 Pertussis 1 Broncholithiasis 2 Uncertain
6	3 "Colds" 1 "Asthmatic Bronchitis" 2 Uncertain	50-60	6	3 "Colds" 1 Syphilis 1 Influenza 1 Pertussis
2	1 Pneumonia 1 Influenza	60-70	2	1 Pneumonia 1 "Colds"
1	1 "Colds"	70-80		
6	2 Influenza 2 "Colds" 2 Uncertain	Uncertain	5	5 Uncertain

COMPLAINTS

The early clinical history is often far from typical. In fact, it may be silent. Only the associated bronchitis, chronic pneumonia or abscess formation, etc., attracts attention to the possibility of the condition in certain cases. As might be expected the chief complaints are cough, expectoration, fetor, hemoptysis and fever. These complaints will be considered separately.

COUGH

Cough in bronchiectasis may be severe, hacking and even persistent. It is sometimes impossible to relieve it by codeine and other such medicaments. Even phrenicectomy may fail to relieve the cough. But the cough in itself is not the complaint which makes the patient with bronchiectasis an abhorrence to himself. It is the foul expectoration.

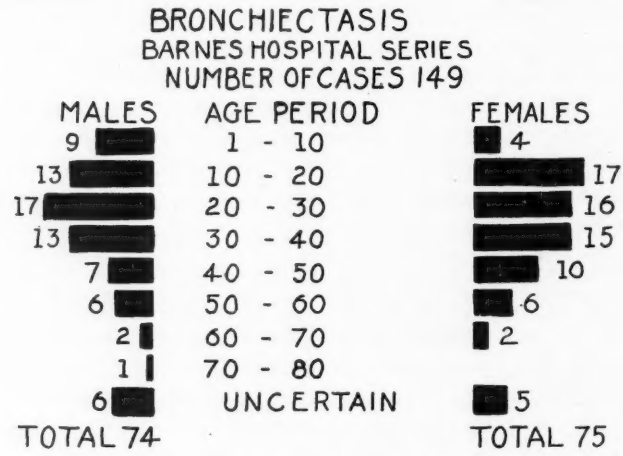

Chart 1.—Represents the sex and age period of 149 patients suffering from bronchiectasis who were observed at the Barnes Hospital Chest Service prior to 1930. (See text for explanation of classification.)

EXPECTORATION

There is nothing particularly characteristic in the composition of the sputum except that it is said to have a higher content of water than the sputum of tuberculosis, there is a considerable amount of albumin, and it usually contains peptones and amino acids. Leucine and tyrosine crystals have been noted. Thissen observed cholesterin and Pyer oxalic acid crystals. There are no typical changes in such inorganic constituents as potassium, sodium and phosphorus.

Dittrich plugs (minute, dirty grayish, ill-smelling masses of bacteria and fatty acid crystals) are usually found in the sputum if it separates out into the three typical layers. Elastic fibers may also occur. Eosinophils are the exception rather than the rule. Fats and fatty acids have also been extracted.

TABLE II

ABSCESS OF THE LUNG PLUS BRONCHIECTASIS
(See text for explanation of classification)
Number of Cases 33

MALE	ETIOLOGY	AGE PERIOD	FEMALE	ETIOLOGY
2	Foreign body	1-10 years		
1	Postpneumonic			
1	Influenza	10-20 years	1	"Colds"
			2	Foreign body
			1	Influenza
			1	Uncertain
2	Posttonsillectomy	20-30 years	1	Influenza
			1	"Colds"
			1	Uncertain
1	Chest injury	30-40 years	1	Tooth extraction
2	Influenza		1	Uncertain
1	Spirochetosis	40-50 years	1	Broncholithiasis
1	Postpneumonic		1	"Colds"
1	Postgastroenterostomy		1	Posttonsillectomy
2	Uncertain			
2	Postpneumonic	50-60 years	2	Postpneumonic
1	Postcholecystectomy			
1	Uncertain	60-70 years		
Total 18			Total 15	

LOBE

Left lower lobe	11
Right lower lobe	18
Right middle and lower lobes	2
Right upper lobe	1
Right upper and middle lobes	1

TABLE III

BRONCHIECTASIS—BARNES HOSPITAL SERIES
(PATIENTS REFERRED TO IN TABLE I.)

Number of Cases	149
Unilateral	83
Bilateral	66

LOBE

Right Upper Lobe	1
Right Upper and Middle Lobes	1
Right Lower Lobe	34
Right Lower and Middle Lobes	5
Left Lower Lobe	40
Left Lower and Upper Lobes	2
Right and Left Lower Lobes	60
Right Lower and Left Lower and Upper Lobes	3
Left Lower and Right Lower and Middle Lobes	2
Left and Right Upper Lobes	1

The amount of sputum is very variable. It may exceed 1,500 c.c. in twenty-four hours. The odor is due to fatty acids, ammonium carbonate, sulphides, indol and other products of putrefaction and autolysis. Ferments are also present.

The sputum may separate out into the following three or four characteristic layers; but the same order is not always followed. The upper layer is whitish or greenish yellow. It is mixed with air bubbles, pus and mucus. The second layer is thinner, contains some of the products of the first layer, and less air. The third layer contains pus cells, detritus, fat rests and the Dittrich plugs.

Bamberger found that the sputum of patients with bronchiectasis contained 93.85 per cent water and 6.14 per cent dry substance. Of this 0.79 per cent of the fresh substance was inorganic, 5.35 per cent organic. Of the dry substance 12.87 parts were inorganic and 71.57 organic.

Lymphocytes do not predominate as in the sputa of tuberculous patients. The yellowish or greenish pus may contain pigment. Yeast cells have also been demonstrated. Bronchial stones and pieces of bone and cartilage have also been expectorated. Reference will later be made to the bacteriologic content of the sputum in bronchiectasis.

HEMOPTYSIS, PULMONARY HEMORRHAGE

Hemoptysis is more frequent in bronchiectasis than it is in tuberculosis. It is often due to bleeding granulations. It may be the first sign to appear and the last to go. It can be alarming, and death from pulmonary hemorrhage in cases of bronchiectasis is not rare. There are certain cases of bronchiectasis described by Bezançon and others in which hemoptysis is the only complaint, the bronchi are otherwise dry. Such patients are not infrequently wrongly considered to be suffering from tuberculosis. Reference will be made to this type again.

FEVER, SWEATS AND CHILLS

Fever, sweats and chills are significant complaints. To the patient they frequently represent repeated attacks of pneumonia. They are indeed attacks of pneumonia (pneumonitis*) with or without abscess formation. When a fibrinous pleurisy does not heal it means that the exudate of fibrin does not become liquefied. Instead, connective tissue proliferation takes place. Such connective tissue cells are ameboid. This connective tissue becomes vascularized in the exudate. New white blood cells come and phagocytose the fibrin. This process lasts for weeks and in the end the vascularized red connective tissue becomes white. When such a process takes place in the alveoli of the lung, then the exudate may be substituted by fibroblasts and capillaries. This connective tissue replacement never disappears. During the early stage when the connective

*The term pneumonitis merely means a nonspecific inflammation of the parenchyma of the lung. It is sometimes used to imply Streptococcus pneumonia. The term has been loosely used by many. Throughout the text, unless qualified, it will be used only in its strict sense.

tissue is red there is produced the picture of carnification. In the late stage the lung becomes gray but never as in healed tuberculosis. Lung tissue so involved no longer functions. Patients so affected later suffer from repeated attacks of pneumonia and bronchiectasis. There is pulling in all directions by scar tissue around the bronchus. An adherent pleura helps in the process of shrinking. The bronchi become filled with secretions and are veritable standing culture tubes. Such a lung on cut surface is smooth because the newly formed connective tissue prevents retraction of the elastic fibers when they are cut. It is thus most important to realize that fever may often be due to a pneumonitis and not merely to retained secretions in the bronchi. Drainage of the latter, therefore, even though adequate, will not always result in the hoped-for reduction of the fever. There is little chance for much parenchymal suppuration when the dilatations of the bronchi are numerous and large. When the dilatations are small and associated with fever and chills, then pneumonitis must be admitted. Paravertebral atelectasis which is associated with bronchiectasis is always significant irrespective of whether it is due to pulmonary fibrosis or bronchial obstruction.

PAIN

Pain is not a striking feature in bronchiectasis. One must consider the possibility of a malignant process, bronchial stone or spontaneous pneumothorax when it is severe.

GASTROINTESTINAL SYMPTOMS

Severe gastrointestinal symptoms in patients with bronchiectasis are usually of ill omen. They are often manifestations of amyloid disease. It is well to remember that some patients appear to vomit their sputum (bronchial vomiting).

SKIN ERUPTIONS

Toxic dermatitis in patients with bronchiectasis is not common and is usually noted during the period when there is lack of drainage. When adequate drainage is established the eruption usually disappears.

JOINT PAIN

Joint pain is not uncommon in bronchiectasis and is but another manifestation of the same process which is responsible for clubbing of the fingers.

DIAGNOSIS

The diagnosis of bronchiectasis should be considered as incomplete unless one is able also to diagnose the location of the disease (i.e., what lobe or lobes are involved), the size and distribution of the dilatations, the degree of fibrosis as evidenced by atelectasis and shift of the mediastinal contents, the nature of the process, whether tuberculous or

not, and the presence or absence of various local or systemic complicating features, such as foreign body, pulmonary abscess, bronchial stenosis, carcinoma, empyema, brain abscess, amyloid disease, etc. It is apparent at once, therefore, that a satisfactory diagnosis of bronchiectasis involves a careful and prolonged examination of the patient. Such an examination often requires the employment of the following sources of information: (1) clinical history, (2) physical findings, (3) laboratory findings, (4) ordinary roentgen-ray findings including fluoroscopy, (5) bronchography, (6) bronchoscopy, (7) diagnostic pneumothorax.

CLINICAL HISTORY

The often repeated statement that a good clinical history is at least half the diagnosis holds true for bronchiectasis. An adequate clinical history should consider the patient's past, personal and family histories in detail. The importance of a previous influenzal infection and of the pneumonias consequent to the exanthemata and pertussis is well known. And in this connection it is well to note that the nature and frequency of previous upper respiratory tract infections may be just as significant as those referable to the lower respiratory tract. It is also well to appreciate that foreign bodies in the lung may be endogenous (bronchial stones) or exogenous. The former may be responsible for just as severe bronchiectasis as the latter. The history should also concern itself with references to the various systems other than the respiratory. Headaches may be an expression of brain abscess and not of toxemia. Cyanosis and dyspnea may be due entirely to myocardial damage, and urinary frequency may be the only indication of severe amyloid disease. One should always find out whether the patient has been exposed to tuberculosis, for although it is true that many patients with bronchiectasis are to be found in sanatoria where they are treated as tuberculous suspects, still it is equally true that one may err in the opposite direction, and the mere demonstration of dilated bronchi does not exclude the possibility of tuberculosis. Bronchiectasis merely means dilated bronchi, a diseased state of the bronchi and so it behooves us to attempt to ascertain the cause whenever possible.

The importance of a good clinical history in the diagnosis of bronchiectasis is obvious; nevertheless at the present time the diagnosis cannot be made on this basis alone. A clinical history suggestive of bronchiectasis must always be substantiated by a positive lipiodol injection picture and sometimes by a bronchoscopic examination. There are occasions when the former may be misleading but in such cases one is misled only as to the extent and degree of the lesion.

PHYSICAL FINDINGS

There are no truly typical physical findings referable to the chest of a patient with bronchiectasis. With a typical history signs referable to

the base are, of course, suggestive. Particularly is this the case when they vary after the patient is postured, that is, when they change after the patient has emptied his lungs of secretions. The signs may be suggestive of areas of atelectasis and of cavitation; there may be increased whisper or diminished breath sounds. Râles are nearly always present.

Hypertrophic osteoarthrophthy was described by Bamberger, 1889, and by Pierre Marie in 1890. Since in most of the articles on bronchiectasis this subject is very briefly discussed, it has seemed advisable in this treatise to present somewhat extensively the newer work on this subject. It has been thought by many that the condition is due to prolonged venous congestion, a peripheral hyperemia being produced by compression of the lung capillaries in pulmonary pleurisy or mediastinal disease. It has also been suggested that hyperemia produces a hyperplasia of the periosteal and connective tissue. Others suggest a toxin. Changes in the nose and jaw draw attention to the pituitary gland and in a few cases changes have been noted in the sella turcica. It is strange, however, that profound changes may take place in the bones without clinical signs or symptoms of disease being present. There may be clubbing without any bone changes. The condition may develop very rapidly and may disappear just as quickly. Locke and others have noted the condition in association with diseases of the respiratory, circulatory and alimentary tracts in cases of syphilis, amyloid disease and disease of the salivary glands.

David Campbell's observations on the hippocratic fingers are most instructive. It is his opinion that edema is the primary pathologic basis in every case of clubbing (Fig. 14-A). He rightly points out that clubbing of the fingers is frequently seen in association with conditions in which there is no obstruction to the venous return from the limbs. The one finding in common seemed to be that a definite, though varying, amount of the lung substance had ceased to fulfill its proper function. Campbell concluded therefore that the diseased area through which the blood flows will not be ventilated, and accordingly the oxygen tension of the whole arterial blood will be lowered. "That this statement is not based on mere theory is shown by the results obtained in experimental work by Stadie in cases of pneumonia and by Haldane in cases where the lungs were damaged by gas poisoning. In both types of disease it was found that the oxygen tension of the blood was definitely lowered—in other words, the patients suffered from anoxemia. It is not suggested that in all the cases under discussion the anoxemia is severe in character; in the majority it is probably only slight, but it is important to recognize that it is present and that its essential feature is its chronicity.

"The transference of oxygen from the blood to the tissues depends upon the difference in tension of the oxygen in the blood and in the tissues. It will be clear that if, from any cause, the arterial oxygen tension be lowered, the tissue respiration will be defective. A small defect in tissue respiration may not produce any demonstrable effects on the individual, but, if it does, these will be seen in parts of the body where the defect is increased by a slower circulation. These parts, owing to mechanical reasons, are the tips of the fingers and toes, and the nose. This is very well illustrated in cases of pneumonia where sometimes cyanosis can be observed under the finger nails before it has made its appearance in any other part of the body."

Campbell believes that this theory is capable of wide application and explains the clubbing so frequently seen in congenital heart disease and also the clubbing in the condition known as autotoxic enterogenous cyanosis. In the latter case there is present in the blood either methemoglobin or sulphemoglobin, the result of the absorption of the products of intestinal putrefaction. These compounds of hemoglobin have little or no power to carry oxygen.

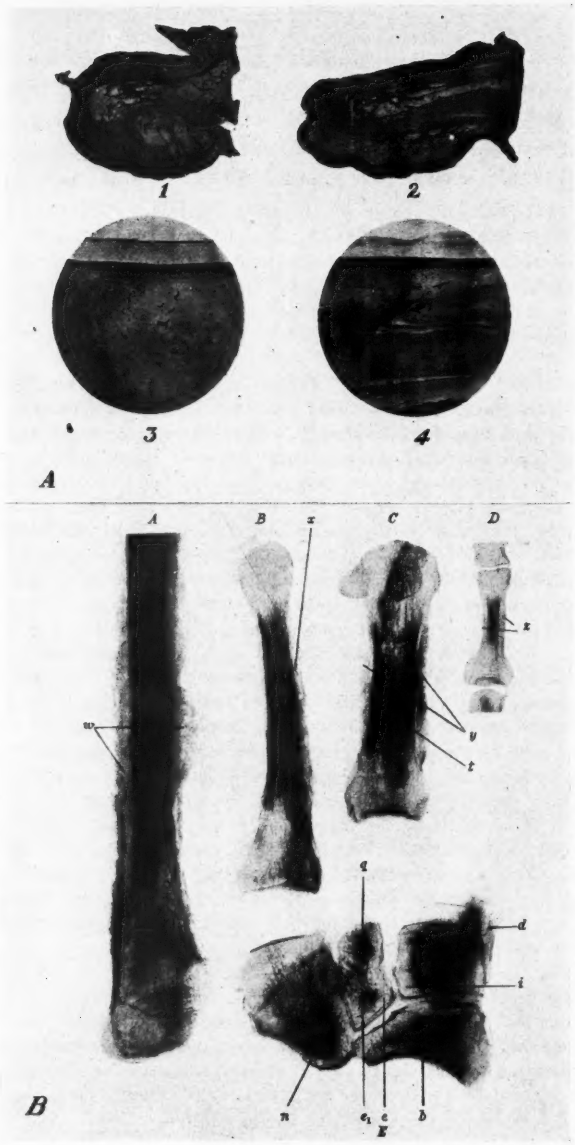

Fig. 14-A.—Hippocratic fingers (after David Campbell: Brit. Med. Jour., January 6, 1924, p. 145). (1) Clubbed finger (×4). (2) Normal gngfier (×4). (3) Nail bed, clubbed finger (×20). (4) Nail bed, normal finger (×20).

Fig. 14-B.—The roentgen ray appearance of various bones in hypertrophic osteoarthropathy (after C. Crump; Virchows Arch., 1929, vol. 271, p. 467).

(*A*) Roentgen ray appearance of the fibula; (*B*) of the metatarsal; (*C* and *D*) of the phalanges; (*E*) of the novicular bone; (*d*) 1st cuneiform; (*i*) 2nd cuneiform; (*q*) 3rd cuneiform; (*n*) os cuboideum.

Thickest portion of the periosteum in the fibula (*w*); its appearance in the dorsal aspect of the metatarsal (*x*); and its appearance on the medial and lateral aspects of the phalanges (*y*) *and* (*z*).

For other details the reader is referred to the original article.

The arguments which tend to show that clubbing is the result of defective oxygenation are certainly worthy of consideration. Farquhar Buzzard in a case reported originally by Walters found no degenerated fibers in the median, ulnar, sciatic and anterior crural nerves.

The most recent and best work on the histology of hypertrophic pulmonary osteoarthrophthy is that published by Crump (Fig. 14-B). He showed that changes may be referable to the bone, the periosteum, and the joint. The changes in the soft parts of the end phalanges are the ones which produce a typical clubbing. Locke has reported the results of roentgen-ray examination in 39 cases of simple clubbing of fingers and toes. The diseases varied and were referable to the pulmonary, circulatory, gastrointestinal and other systems. As is usually the case, changes in the distal phalanges were noted only on five occasions, changes in the long bones on twelve occasions. Roentgenologic changes in the phalanges are far from common. The articles by Locke and Crump contain a most complete bibliography.

LABORATORY FINDINGS

The blood count may show a moderate anemia and, as might be expected, a varying leucocytosis. It was a striking fact that few of the patients who presented themselves at Barnes Hospital had a positive Wassermann reaction. Syphilis was more frequently observed in association with pulmonary tuberculosis. Reference has already been made to the nature of the sputum in bronchiectasis.

The presence of albumin in the urine should always suggest the possibility of amyloid disease. We have been impressed with the fact that a normal blood nonprotein nitrogen, and only moderate albuminuria do not preclude the possibility of severe amyloid disease. Of significance is perhaps the fact that the duration of the suppuration in such cases is usually of many years. A slight operation, even one which promotes adequate drainage of the suppurative focus, may be sufficient to precipitate a fatal uremia as we observed in a recent case.

BACTERIOLOGIC FINDINGS

Bronchiectasis is a disease without specific microorganisms. D. T. Smith, however, considers a spirochete to be a definite etiologic factor in certain types of so-called primary bronchiectasis. We need not consider this point further, here, since it has been referred to in detail in the discussion on the relationship of fusospirochetal disease to bronchiectasis (see previous chapter). Steinmeyer and Kathe examined the sputum of ten patients suffering from bronchiectasis and found the following organisms:

Streptococcus viridans, 6 times; Streptococcus hemolyticus, 4 times; Streptococcus nonhemolyticus, 1 time; Pneumostreptococcus, 1 time; Staphylococcus aureus hemolyticus, 4 times; Bacterium Coli commune, 2 times; Bacterium Coli Hemolyticum, 2 times; Bacillus of the paratyphoid group, 1 time; Leptothrix, 3 times.

Robinson and Greey reported the bacteriologic findings in eight cases of bronchiectasis in which a lobe or a portion thereof was removed at operation and sent immediately to the laboratory for culture. "Direct smears, dark-field preparations, aerobic and anaerobic cultures of various types as well as bacterial stains and silver preparations of sections revealed nothing consistent enough to be pointed to as a specific infective agent. Cultures were taken from the exudate in the lumen and also from scrapings of the wall. No common flora were recovered. Three cases yielded pure cultures of streptococci, of hemolytic, nonhemolytic and viridans types, re-

spectively. Two showed mixed aerobic cultures. One other case gave a mixture of pneumococcus, Type IV, with spirochetes and fusiform bacilli. In only three of the eight cases were spirochetes found.''

The studies of Lucille Ermatinger are most instructive. Her investigation concerned patients from the Chest Service at the Barnes Hospital. She studied the bacteriologic findings in the sputum in seven cases of pulmonary abscesses. Her findings were as follows:

Predisposing Factor	*Microorganisms*
Pertussis and measles	Staphylococcus aureus, Streptococcus viridans, Micrococcus catarrhalis
Foreign body (blade of grass)	Staphylococcus aureus, Hemolytic streptococcus, Micrococcus catarrhalis
Pneumonia	Staphylococcus aureus, B. xerosis, Micrococcus catarrhalis
Foreign body (broncholithiasis)	Hemolytic streptococcus, B. mucosus-capsulatus
Tonsillectomy	Staphylococcus aureus, Hemolytic streptococcus, Micrococcus catarrhalis
Postoperative (gastroenterostomy)	Staphylococcus aureus, B. mucosus-capsulatus, Micrococcus catarrhalis
Pneumonia	Staphylococcus aureus, Hemolytic streptococcus, B. mucosus-capsulatus, Micrococcus catarrhalis

This work was carried out before that by Varney and there is therefore no reference to the bacillus melaninogenicum which is discussed later in connection with his work.

Ermatinger's findings in eleven cases of bronchiectasis were as follows: (The sputum was examined in all cases and direct wound cultures were taken in all cases involving operation.)

Predisposing Factor	*Microorganisms*
Foreign body (broncholithiasis)	Staphylococcus aureus, Micrococcus catarrhalis, B. mucosus-capsulatus
Influenza	Staphylococcus aureus, Hemolytic streptococcus, Micrococcus catarrhalis
Pneumonia	Staphylococcus aureus, Pneumococcus, Micrococcus catarrhalis, B. mucosus-capsulatus, B. xerosis
Influenza	Streptococcus viridans, Pneumococcus, Micrococcus catarrhalis
Pertussis and measles	Hemolytic streptococcus, Micrococcus catarrhalis, B. xerosis
Pneumonia	Hemolytic streptococcus, Micrococcus catarrhalis, Staphylococcus aureus
Bronchitis	Streptococcus viridans, Pneumococcus, Micrococcus catarrhalis
Pertussis and measles	Hemolytic streptococcus, B. mucosus-capsulatus
Pertussis and measles	Hemolytic streptococcus, B. mucosus-capsulatus, B. xerosis, Micrococcus catarrhalis, fusiform bacillus
Pertussis	Staphylococcus aureus, B. mucosus-capsulatus, Micrococcus catarrhalis
Tonsillectomy	Staphylococcus aureus, Pneumococcus

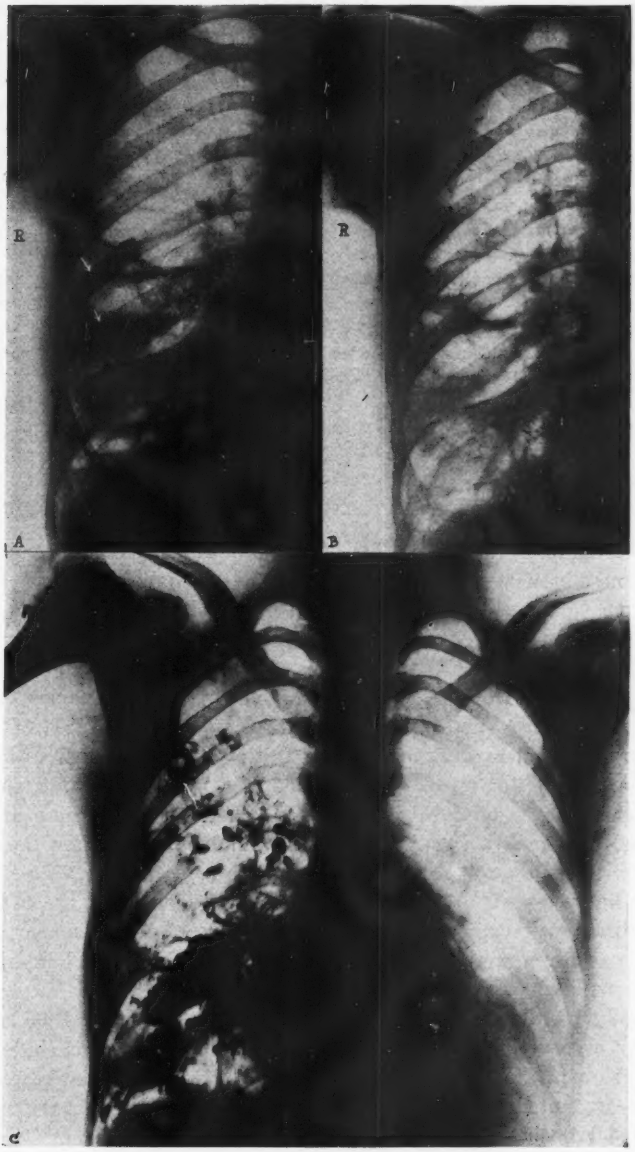

Fig. 15.—*A*. Saccular bronchiectasis in all probability of the congenital type. This occurred in a female, fourteen years of age, who was considered to be suffering from congenital syphilis (?). The arrows indicate the fluid levels. This film was taken before postural drainage. Compare this figure with 15-*B* which was taken after postural drainage. It shows that the dilatations are relatively empty. *C*—represents the lipiodol injection picture in this case. The iodized oil was introduced bronchoscopically by Dr. Arbuckle. This patient also had definite bronchiectasis in the left lower lobe. It was apparently of the acquired type.

It is seen from Ermatinger's work that the Staphylococcus aureus was found in 75 per cent of the cases. It is impossible to conclude just what part the microorganisms found played in the origin of the bronchiectasis.

Varney later also investigated the bacterial flora of treated and untreated lung abscesses in patients observed at the Chest Service of Barnes Hospital. Many of these patients also had an associated bronchiectasis. His work is of special importance because for the first time it implicates as a conspicuous factor in pulmonary suppuration the bacillus melaninogenicum which is a minute strikingly anaerobic nonmotile pleomorphic gram-negative rod. It measures 0.8 by 1 to 3 microns. When grown under anaerobic conditions on media containing hemoglobin after an extended period of incubation, intensely black colonies form which rapidly remove the last vestige of hemoglobin from the plate, leaving it entirely colorless similar to a plain agar plate. The organism was given the above name because the pigment was believed to be melanin (Varney). Varney found that the streptococcus, fusiform bacilli, bacillus melaninogenicum and spirochetes were the organisms most commonly encountered in the sputum of untreated patients. In the treated patients the fusiform bacilli, spirochetes and bacillus melaninogenicum greatly decreased in number or disappeared altogether coincidentally with a relative increase in streptococcus hemolyticus (Varney).

ROENTGEN RAY FINDINGS: FLUOROSCOPY

The ordinary roentgen ray film of the chest in many cases permits one to diagnose bronchiectasis but, as already noted, no diagnosis is complete without a lipiodol injection. Increased hilus detail which extends down to the base and "circular" shadows (Fig. 15) are very suggestive of bronchiectasis, although the former may be only the branches of the pulmonary vessels. Fluoroscopic examination frequently enables one to discover that such increased detail is associated with an area of paravertebral atelectasis or chronic mediastinitis which extends from behind the heart. The fluoroscopic examination is of particular value, since it tells whether the diaphragms are fixed (whether or not there are adhesions), the condition of the mediastinum and whether or not the heart shifts with change of position.

The fluoroscope is also of value in helping to determine the presence of inspiratory or expiratory dyspnea. Such dyspnea is frequently met with in children, and is apparently associated with limited intercostal movement and with impaired diaphragmatic function. Expiratory dyspnea with the diaphragm occupying a deep or low position is frequently observed in children suffering from capillary bronchitis. In them the expiratory dyspnea and deep position of the diaphragm are partly accounted for by the edema of the mucous membrane of the terminal bronchioles, and by retention of secretion in them. As Rach and others have shown, this combination of edema and retention of secretion results in alveolar collapse and this in turn puts a greater load on the diaphragms. We have observed this phenomenon repeatedly, particularly in children suffering from bronchiolitis and bronchiolectasis.

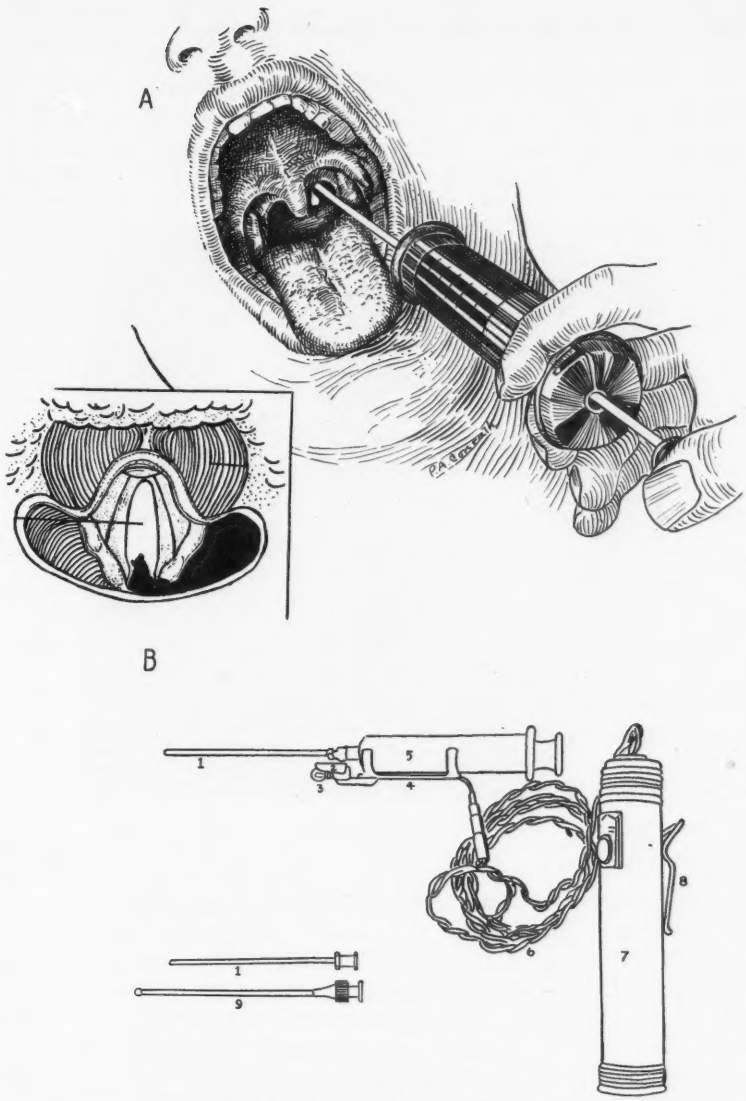

Fig. 16.—Aspiration method of injecting lipiodol: J. J. Singer. *A*.—Method of introducing the iodized oil. The patient faces a good light and is told to open his mouth as widely as possible; his tongue is grasped with a piece of gauze and is pulled out as far as possible. Iodized oil which has previously been warmed in a syringe is slowly injected into the posterior pharynx through a straight cannula while the patient breathes deeply. The patient is urged not to swallow or cough during the injection. The use of a local anesthetic is seldom necessary.

The insert in *A* shows how the iodized oil collects in the interarytenoid fossae. It then trickles between the vocal cords during the phases of respiration and is thus aspirated into the bronchial tree.

B.—A light attachment for syringe (Ballon); this entire instrument is made by Wm. A. Phillips, 922 Pine Street, St. Louis, Missouri. (*1*) cannula; (*2*) removable hood, over light bulb (*3, 4*); (5) 20 c.c. "Lok" syringe; (6) coil; and (7) battery of the ordinary flash-light type; (8) hook by means of which battery can be attached to pocket or hung on the wall; (9) is another variety of cannula. The light is of the ordinary inexpensive type and with it one can perform the injection in a fluoroscopic room.

BRONCHOGRAPHY: METHODS OF INJECTION

It is now possible to inject iodized oil into the bronchial tree by many methods. Unfortunately some of the simpler methods are not satisfactory and cannot be carried out in children. It is, therefore, advisable

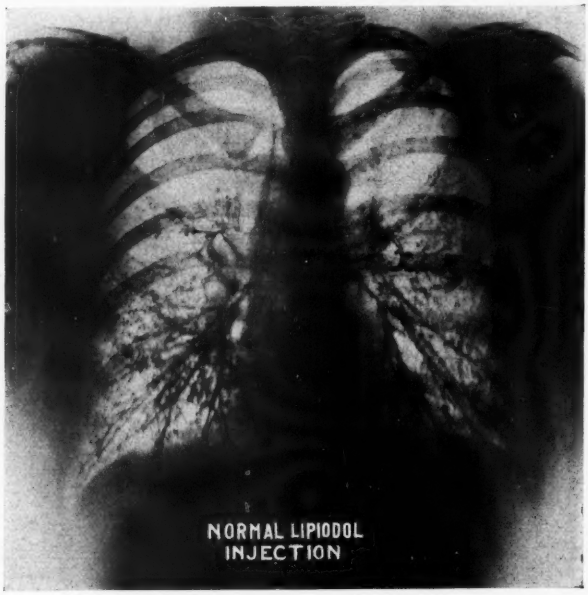

Fig. 17.—Normal lipiodol injection picture (aspiration method).

to be familiar with at least two methods of injection, and for that reason we have recorded the various methods which are in practice now.

I. Supraglottic:
   a. Aspiration method (Singer, Fig. 16)
   b. Supraglottic method (Ochsner)

II. Transglottic:
   a. Catheter (Sgalitzer, Lorey and others)
   b. Intubation tube (Iglauer)
   c. Bronchoscopic (D. H. Ballon and others)

III. Subglottic (Transcutaneous):
   a. Tracheal puncture, intercricothyroid (Armand-Delille and others)

SOME OF THE REASONS WHY BRONCHOGRAPHIC METHODS ARE OF VALUE

The wall of a normal bronchus of the primary and secondary divisions of the bronchial tree does not cast a roentgen ray shadow. The diseased bronchus does cast a roentgen ray shadow but under ordinary circum-

stances it is extremely difficult to determine whether increased hilus detail concerns structures other than the bronchial tree. For this and other obvious reasons it is important to inject lipiodol into the bronchial tree to verify the presence of dilatation which an ordinary roentgen ray film may suggest (Fig. 17). Ballon and Ballon previously reported a classification of bronchiectasis which illustrated five main radiologic types as demonstrated by lipiodol (Figs. 18 and 19). It has since been possible to correlate some of these radiologic types with the underlying pathologic lesion. These will be discussed later. It is all important that more observations be made concerning the future of some of these forms of bronchiectasis when untreated. Ballon and Ballon in 1927 also re-

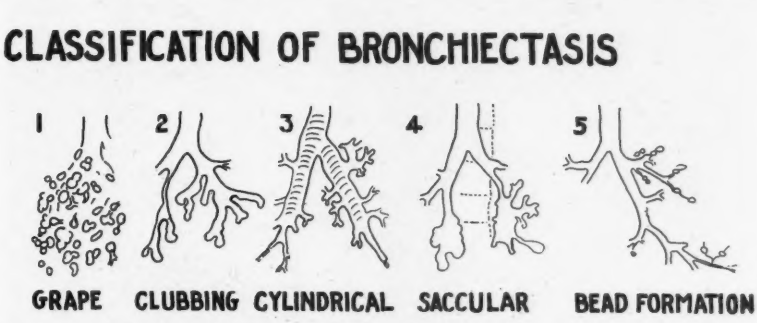

Fig. 18.—The above classification of bronchiectasis by Ballon and Ballon based on bronchoscopic lipiodol injections represents types most commonly encountered. Some of them in all probability represent various degrees of bronchiectasis. More than one type may be present in the same lung. (Reproduced from Arch. Surg. 1927, p. 184.)

corded some observations on the rate of disappearance of lipiodol from normal and from the diseased lungs. It was then shown that posture hastened the disappearance of the lipiodol of the bronchi and that cough could also help. Reference was also made to the lipolytic activity of the lung. It was then pointed out that the fine terminal bronchiectatic dilatations often fail to fill immediately upon injection and bronchiectatic dilatations could empty themselves very quickly but that they might also retain lipiodol for a considerable period. That the shape and position of dilated bronchi varied with inspiration and expiration recorded on the same film was also shown by the two above-mentioned investigators.

THE EMPTYING CAPACITY OF DILATED BRONCHI

Jacobaeus, Selander and Westermark have carried out some studies on the emptying capacity of the bronchi. They employed lipiodol which was stained red with Sudan III. We have shown experimentally that there is no reaction to the latter substance. Jacobaeus and his coworkers believed that they could distinguish two phases in the expectoration. It is believed that the first phase is an expression of the emptying capacity

of the bronchi and that the second phase corresponds with the emptying of the finest bronchioles and alveoli. The second phase is thus much slower and more incomplete than the first. It was their opinion that the primary phase under normal conditions lasted from four to eight hours and that it was followed by a transitional period of eight to twelve hours

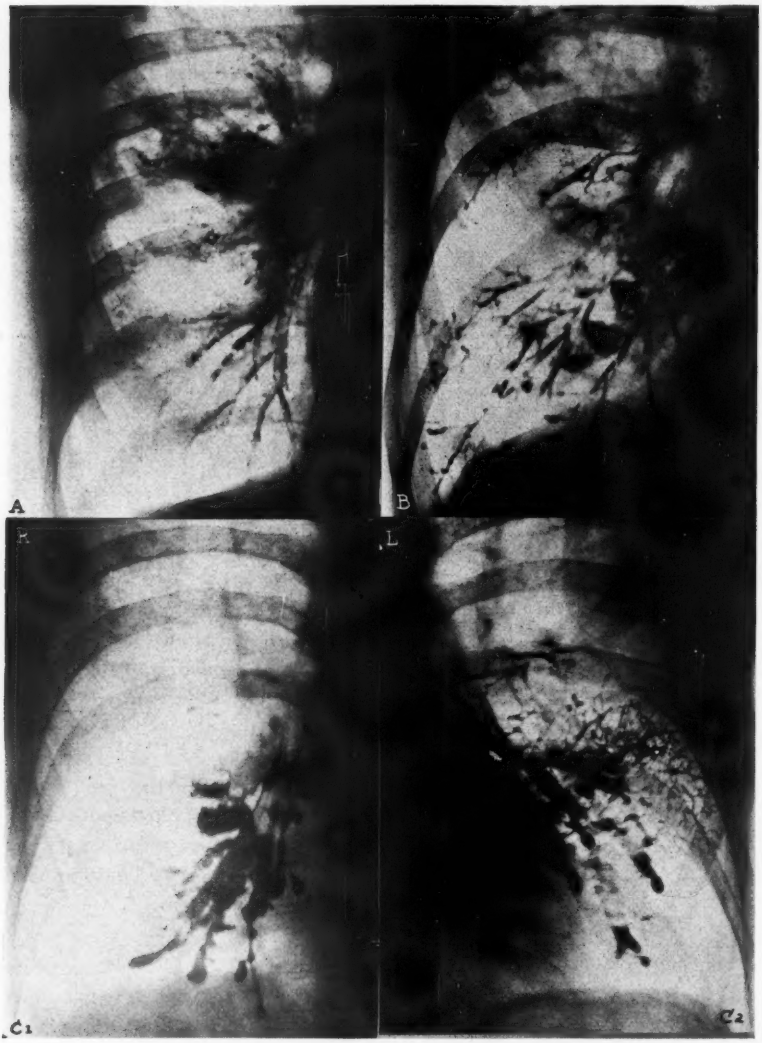

Fig. 19.—*A*. Note the dilatations extending from the hilus. These are of the saccular variety and are probably congenital. Saccular dilatations of the acquired type are illustrated in 19-B. Note the number of normal bronchi which surround these dilatations. 15-C1 and 15-C2 represent two types of bronchiectasis which were observed in the same patient, the latter is of the atelectatic type.

during which time globules of lipiodol were still coughed up. This stage is said to be entirely lacking often, while the second stage may be characterized by the presence of streaks or spots of lipiodol in the sputum frequently observed over a period of several days without apparently following any obvious rule. Unfortunately Jacobaeus and his coworkers did not have true normal cases. The expectoration time in the so-called normal cases was found to be four to eight hours.

We have repeated some of these observations and again verified the fact that the lipiodol injection unfortunately fails to give a complete picture of the underlying lesion. Thus, one cannot conclude that those bronchi which are injected with lipiodol are necessarily those from which all the sputum is coming.

For example, if one injects some dilated bronchi, it is possible to observe these injected bronchi for several days without the loss of any stained lipiodol although, in the meantime, there has been an abundant expectoration. Again, the lipiodol may only sink to the bottom of certain dilatations after several days. This immediately implies that the original observation does not permit one to conclude just what lipiodol is in the alveoli, or whether it will eventually enter the alveoli. It is, of course, possible but unlikely that lipiodol may merely coat the bronchus and that unstained expectoration may come through such a coated bronchus. It has been found that the expectoration time may vary in the same individual. This variance is to some extent dependent upon the amount of oil injected and upon the type of dilatation. And so in our experiences it has not been possible to obtain any trustworthy observations about the expectoration time since there are so many variable factors. These variable factors concern such matters as whether or not there is much fibrosis, atelectasis, mediastinitis, elevation or fixation of the diaphragm.

One can assume the presence of an associated bronchiectasis in those patients in whom the physical signs or roentgen ray suggest something more than an abscess. This also holds true for patients who do not progress well after an operation for abscess of the lung; for, as one of us (H. C. B.) pointed out and illustrated by lipiodol injections several years ago, chronic abscesses of the lung are always associated with some degree of bronchiectasis. It is usually impossible to obtain a positive injection picture in such a case till after the abscess is drained. But if one injects the sinus, then the presence of the associated bronchiectasis can readily be demonstrated.

BRONCHOSCOPY

The bronchoscope is a great aid in the diagnosis of bronchiectasis for the following reasons:

1. It enables one to rule out the presence of a foreign body, new growth or bronchostenosis.

2. It permits one to observe which bronchi are discharging pus.

3. It allows of the most complete lipiodol injection since it is possible to aspirate secretions and remove granulations before introducing the oil. By this method one can also direct the lipiodol to the desired area. Most important of all, it permits of a more exact interpretation of the injection picture. It also allows one to exclude many of the causes of such an unexplained symptom as hemoptysis.

4. It is possible to obtain uncontaminated cultures by this method.

The value of bronchoscopy in the treatment of bronchiectasis will be referred to elsewhere.

DIAGNOSTIC PNEUMOTHORAX

The value of diagnostic pneumothorax in a suspected case of bronchiectasis has been discussed by Singer. In 1926 he stated, if sufficient air is introduced, then a bronchiectatic atelectatic lobe which is so often hidden in the cardiac shadow may be brought out of the diaphragmatic sulcus and is rendered visible in the roentgen ray film. Since lipiodol is able to outline such hidden dilatations there is seldom need for this procedure.

THE RELATIONSHIP OF NASAL SINUS DISEASE TO BRONCHIECTASIS

The fact that a relationship exists between disease of the nasal sinuses and pulmonary suppuration is of comparatively recent knowledge, and its importance is just now being recognized. Because of the widespread interest which is being taken in this relationship it has seemed advisable to present a detailed discussion of the subject.

Lymphatic absorption from the antrum and frontal sinus, whether bacterial or of inert substances, is by the way of the submaxillary and internal jugular nodes to the lymph ducts, the great veins, the right heart and the lungs (W. B. Mullin). And in respect to lymphatic function as well as anatomically, it has been stated that the lining membrane of the nasal fossae and accessory sinuses is a continuous whole. Infection at one point can probably be conveyed by lymphatics to practically any other point, but in the case of the frontal sinus aspiration via injured veins is not excluded. However, Mullin and Ryder were unable to demonstrate a direct lymphatic communication from the plexuses and nodes of the head and neck to the lungs and pleura.

Sergent was perhaps the first to note the association of nasal sinus disorders and respiratory disease. It should be noted, however, that Sir St. Clair Thomson in 1914 drew attention to the fact that the inveteracy of bronchorrea might be explained upon the basis of nasal sinus suppuration. Graham and Singer were among the first to draw atten-

tion to the association of nasal sinus infection and bronchiectasis. In a discussion on the surgical treatment of bronchiectasis one of us (E. A. G.) in 1925 made the statement that "somewhat allied to measures directed against the exciting cause is the matter of correcting suppurations in the nasal sinuses. Astonishing improvement, especially in the cases of chronic pulmonary suppuration, often follows the treatment of chronic nasal suppuration." Raffo believes that sinusitis and bronchiectasis are frequently of simultaneous origin; that once both are in existence they exert an influence on one another. "This is especially true in the sense that sinusitis affects bronchiectasis. The treatment of bronchiectasis is strengthened by treating the sinus at the same time." Raffo reports examples.

Rist considered the condition of the upper respiratory tract of importance in diseases of the lungs and in the differential diagnosis of pulmonary conditions. In respiratory disease, he writes, the whole upper respiratory tract must always be examined, for an examination of the urinary tract is not confined to the urethra, prostate or bladder alone and from the standpoint of the investigation of disease the examination of both does not essentially differ.

Adam raises the question as to whether repeated inspiration of pus from the antrum, especially during sleep and in patients subject to recurrent bronchitis, might not also be a factor in producing bronchiectasis. He reports four cases.

Mullin believes that accessory nasal sinus disease and cough occur simultaneously during childhood, that the advanced cases have pansinusitis, and that the bacterial flora from the sinuses and sputa are the same. He notes further that a goodly number of patients have no symptoms or clinical evidence of sinus disease such as is usually manifested by discharge and headache, but that the sinuses are nevertheless diseased. This was apparently found to be especially true when the maxillary sinuses were concerned.

Davis is of the opinion that nasal sepsis can influence the lower respiratory tract in three ways: (1) by direct extension of the inflammation to the larynx, trachea or bronchi, or, by infection through the blood and lymphatic streams, (2) by aggravating or increasing an existing lesion of the lungs, and (3) the nasal sepsis may be part of the general infection in which both the upper and lower respiratory tracts are involved.

Graham's observations on the reaction of bronchial fistulas to acute infections of the respiratory tract are interesting in this connection. He found that a frequent phenomenon in those patients who have bronchial fistulas is the occurrence of an acute inflammation of the bronchial mucous membrane in the presence of an acute infection of the upper respiratory tract. Under ordinary conditions the discharge of mucus secreted from a bronchial fistula is slight but when a patient

with such a fistula develops an acute infection of the nasal sinuses, then the mucous membrane of the exposed bronchi becomes red and swollen and discharges large quantities of secretion which is at first almost pure mucus but which later becomes mucopurulent. "Local manifestations of acute infection in the bronchus usually do not appear until about twenty-four hours after the onset of the acute sinus disease. With the subsidence of the acute infection in the nasal sinuses the local reaction in the bronchus also subsides. The mucous membrane loses its swelling, assumes a more nearly normal color and the amount of secretion of mucus greatly diminishes. Bacteriologic examinations of the secretion from the nasal sinuses and from the exposed bronchus almost always reveal the same organism. In the cases in which the examination was made the prevailing organism has been streptococcus."

In discussing naso-oral sepsis Young states that there are at least six ways in which local septic disease in the mouth, nose and pharynx may affect the respiratory mechanism: (1) by direct spread of infection; (2) mechanically (nasal obstruction is present and prevents a proper filtration and warming of the air, and this in turn promotes catarrhal inflammation of the alveoli; (3) from the effect of inhalation, (a) of septic organisms from the nose and mouth, (b) of masses of infective material; (4) by altered blood and immunity conditions with absorption from foci from these diseased areas; (5) when oral sepsis leads to septicemia; (6) by causing myositis, pleurodynia, etc.

The influence of inspirated infectious material on the mucosa of the larynx, trachea and bronchi has frequently been observed at the Jackson Clinic. Clerf notes that mirror laryngoscopy frequently reveals a chronic laryngotracheitis in patients who complain of cough and huskiness of voice and in whom thorough physical examination and x-ray study of the chest show no evidence of disease. Investigation of the paranasal sinuses of such patients revealed the presence of chronic infection. Clerf further notes that bronchial dilatation cannot be demonstrated by bronchoscopy or by pneumonography in certain instances, but that changes are observed about the carina and bronchial spurs. Such changes usually consist of widening of the angle of division of the trachea or bronchi, the carina and spurs being broad and round. These changes are interpreted as evidence (Mullin) of enlargement of the peritracheobronchial and peribronchial lymph nodes. The relationship between chronic infection of the accessory sinuses, laryngotracheitis, bronchitis and enlarged peribronchial glands is thus seen. As chronic bronchitis is not infrequently a forerunner of bronchiectasis, the direct connection that exists between nasal sinus infection and bronchiectasis becomes fairly obvious in certain instances at any rate. There are undoubtedly occasions when the nasal sinuses and lower respiratory tract are simultaneously infected. This is particularly true of some of those cases which are due to influenza.

Clerf attempted a differentiation between cases of bronchiectasis commonly associated with diseases of the nasal accessory sinuses and those which are unassociated with nasal sinus disease. The latter group includes examples of bronchiectasis due to noninfluenzal pneumonias, abscess of the lung and foreign body in the lung. The former group particularly includes those cases due to influenza or cough which date back to early childhood. Some of the patients who fall into the second group may also have nasal sinus disease. Clerf raises the question as to whether patients with chronic infection of the nasal sinuses are to be considered potential bronchiectatics. He presents a case which suggests this possibility and in concluding emphasizes the fact that a negative opinion on sinus disease should not be given until every necessary diagnostic means has been utilized. Chronic cough in children demands such investigation and prompt treatment, for by so doing undoubtedly some patients with early bronchiectasis will be improved and even potentially cured.

Since bronchiectasis is often a sequela of influenza it is interesting to find that numerous pathologists have called attention to the frequency of nasal sinus disease in influenza. It does not necessarily follow, however, that in these cases the bronchiectasis is produced as a result of the sinus involvement, but at least the observation of the frequency of sinus lesions in influenza is important.

Involvement of the nose and of the sinuses has long been considered a characteristic symptom of influenza according to Lucksch who has made a pathologic anatomic study of influenza based on German sources. Weichselbaum was apparently among the first to make anatopathologic studies along such lines. Inflammation of the accessory sinuses of the nose develops later (Lucksch). Inflammation occurs most frequently in the sphenoidal sinus, in the other sinuses or the orbit it is more rare and it usually ends in suppuration. The hemorrhagic form is also observed alone or associated with the suppurative form, that is with empyema of the sinuses. The influenza bacillus and a filtrable virus were found in the nasal secretion. The former was associated with various cocci, chiefly Micrococcus catarrhalis. In the sinuses the influenza bacillus is frequently associated with the streptococcus.

Opie, in a study of the pathologic anatomy of influenza, based chiefly on British and American sources, states the following: "Infection of the accessory nasal sinuses appears to be an almost invariable accompaniment of influenzal pneumonia. In twenty examinations made by Abrahams, Hallows and French they were found to be normal once. Bright yellow pus usually filled the sphenoidal sinus, but occasionally there was turbid serous fluid and the membrane was deep red. In twenty autopsies on those who had died following influenza, Wolbach examined the sinuses and found infection in the sphenoidal sinus in all, in the frontal sinus in seven and in the ethmoidal sinus in eight. In-

fection of the middle ear occurred thirteen times. B. influenza with pneumococcus or streptococcus were grown from the contents of the sinuses. Similar observations have been made by Stone and Swift and by Lucke, Wright and Kime."

The severity of a sinusitis depends upon the power of the mucosa to prevent its destruction by the inspired microorganisms. A progressively increasing poisoning of the ciliated epithelia by the toxins occurs, and as this ciliary paralysis increases in amount the secretion of mucus is also paralyzed. Phillips who is responsible for the above statement is convinced that a healing ciliated epithelium is a most important factor in maintaining a normal sinus condition.

Dunham and Skavlem found that 28 per cent of a total of 389 patients observed with suspected respiratory disease during a period of two years were suffering from foci of infection other than the lung. Dunham was of the opinion that almost every case of purulent bronchitis or apical catarrh which had come under his observation had been accompanied by sinus infection. In a series of twenty-five cases of bronchiectasis eight were "secondary" to infected antrums, four to ethmoids and thirteen to infected antrums and ethmoids.

Cone has presented a case of bronchiectasis associated with paranasal sinus disease in a child, ten years of age. The bronchiectatic cavity in the right base was treated bronchoscopically and by postural drainage, the nasal condition by irrigation; both with appreciable improvement.

Dennis has published an instructive study which embraces 50 cases of accessory nasal sinus infection with complications. The diagnosis in 24 instances was bronchiectasis, in 8 asthma, while another group of 8 included nephritis, lung abscess, cardiac involvement and arthritis. The following is an analysis of those cases which concerned nontuberculous pulmonary lesions. Ten patients dated their trouble from an attack of influenza. In eleven the treatment consisted of puncturing and washing the antra; 44 patients had radical antrum operations, 19 of which were bilateral. In eight the ethmoids only were operated on and in 17 the sphenoid was opened together with other sinuses. In practically all cases with bronchiectasis or asthma the maxillary sinus was diseased (47 out of 52 cases). Seventeen of the 24 patients suffering from bronchiectasis and nasal sinus disease were examined before they had reached the fortieth year. Twenty of these patients had contracted their disease before the age of 40. Pus was noted in the sinuses in 28 of the 60 patients examined and polypoid degeneration in 45 instances. In the 52 cases of bronchiectasis and asthma pus was found only thirteen times. In the vast majority of instances there were hyperplastic changes in the mucosa.

The rôle of the accessory sinuses in nontuberculous pulmonary infections from the standpoint of diagnosis and compensation has been dis-

cussed by Malone of the United States Veterans' Bureau. He has noted that chronic sinusitis, especially when complicated with some form of nontuberculous pulmonary involvement, is presenting a problem of increasing magnitude to the Veterans' Bureau. This author divides all cases into the following five classes. (1) Sinus infection with no manifest pulmonary lesion but with definite constitutional symptoms; (2) sinus infection with a mild degree of lung complication, a chronic bronchitis; (3) sinus infection with a more extensive pulmonary involvement, an interstitial pneumonia; (4) sinus infection with bronchiectasis; and (5) sinus infection complicated with lung abscess. Malone found that the maxillary sinus was the chief offender and that the lesion most frequently met with a thickened mucosa.

Daland has noted that systemic disease secondary to chronic infection of the tonsils or sinuses is frequently associated with leucopenia, lymphocytosis and diminished polymorphonuclear cells in about 40 per cent of cases.

Quinn and Meyer have observed patients who were treated for tuberculosis over a period of years and who in reality were suffering from sinusitis and bronchiectasis. In some instances the sinuses had not even been examined. The association of nasal sinus disease and nontuberculous pulmonary suppuration may perhaps be more frequent than the association of sinus disease and pulmonary tuberculosis. Quinn and Meyer found the association of sinusitis and bronchiectasis to be less frequent than previously reported. They observed it in twenty-one of the thirty-eight patients examined. The average duration of symptoms was 5.7 years. No direct relationship was determined between the degree of sinusitis and the degree of bronchiectasis. In fifteen instances, tonsillectomies had been done. Sixteen of the thirty-eight patients had no evidence of sinusitis. They have noted that patients with bronchiectasis and sinusitis were considerably younger than those with bronchiectasis and without sinusitis. The majority of their patients had no symptoms of sinusitis. Quinn and Meyer have been able to show among others that aspiration of substances, such as iodized oil, into the lung from the nasal fossae while one is asleep is possible and does occur with apparent ease and that not infrequently the quantity aspirated is surprisingly large.

Webb and Gilbert have rightly pointed out that the internist must first suspect the nasal or sinus disease which may be associated with bronchiectasis and bronchitis. They report 37 cases in which findings were confirmed by opening and washing of pus from the sinuses. Such pus usually contained pneumococci similar to those found in the sputum.

The relation of deficiency diet to disease of the sinuses has been discussed by Dean, Armstrong, Hutton and others. Experimentally it is possible to produce bronchiectasis in guinea pigs by feeding them a diet deficient in vitamines (Goldblatt and Benichek).

It is worthy of note that inflammations and suppurative processes in the paranasal sinuses are known in infants only a few weeks old (Paunz) and good radiographs of paranasal sinuses may be had in infants and are quite accurate in their portrayal of the anatomy and pathology (Wasson). An extensive bacteriologic study of nasal sinus disease in infants and young children has been made by Dean and Armstrong.

We now present our own experiences in a series of 37 cases of nasal sinus infection and bronchiectasis. Many more are available, but these 37 cases seemed to be enough to make the point (Fig. 20). The etiology of the bronchiectasis was influenza in only six instances. Seven of the

Fig. 20.—Represents the type of bronchiectasis frequently observed in patients who are also found to be suffering from nasal sinus infection. The respiratory symptoms of some of these patients are definitely improved following treatment of the nasal sinuses but the dilatations remain unaltered.

patients had pansinusitis. The maxillary sinus was the chief offender. In some instances the sinuses were drained, in others radical operations were performed. The usual findings were a thickened mucous membrane and granulation tissue. The following cases are illustrative of the association of sinusitis and bronchiectasis and of the results that are to be expected. Note the numerous associated treatments.

CASE 1.—Bronchiectasis (bilateral): nasal sinusitis.

Treatment: Bronchoscopy, neoarsphenamine injections, pneumothorax (right), phrenicectomy (right), incision and drainage of maxillary sinuses.

T. M., female, aged eighteen, was admitted to Barnes Hospital, January 16, 1927, complaining of cough and foul expectoration of more than ten years' duration. Family history: negative. Personal history: measles, mumps, whooping cough and

chickenpox; influenza three times in seven years; chorea, tonsillectomy several years previous to admission.

The complete physical examination revealed bilateral bronchiectasis. The sputum which varied from one to 380 c.c. in twenty-four hours was repeatedly negative for tubercle bacilli. Bronchoscopic examination was performed by Dr. A. Proetz on January 22, 1927. He observed pus coming from the main bronchi of both lower lobes. The patient's metabolic rate taken during the month of January was found to be plus 65. The general condition of the patient seemed somewhat improved following bronchoscopy. She was then given injections of neoarsphenamine. Lugol's solution was also administered and the patient was discharged on February 8, 1927, moderately improved.

Dr. Varney's report on this patient's sputum was as follows: Large number of degenerated neutrophiles and transitionals. Granular fibrin masses. Very few bacteria in preparation. No spirochetes present. May be some fusiform bacilli present, though this cannot be ascertained for a certainty by microscopic methods. A few gram-positive diplococci are present. Pneumococci constitute a fair percentage of the total bacteria present.

A concentration of the sputum according to the method of Raphael and Eldridge was performed, and a Ziehl Neelsen stain made. No acid fast bacilli were present. (This method gives about 30 per cent more positives than the antiformin method.)

Cultural: Aerobic—hemolytic, Gram amphophilic, pleomorphic coccus or short bacillus present growing in small, irregular colonies. Gram: diplococcus, similar to M. catarrhalis, growing in smooth, gray, entire colonies; no action on blood. Anaerobic—relatively large number of fusiform bacilli present; these are of two types. Gram-positive diplococcus, producing small, irregular, spreading colony; produces green coloration on blood; this is probably the same organism described under the aerobic colonies. Better growth results anaerobically. Very small gram-positive diplococcus or short streptococcus, producing hemolysis of blood, colonies 1 mm. in diameter; rather numerous. It is not a typical streptococcus.

The patient was readmitted on February 25, 1927. Pneumothorax treatment was started and was continued at weekly intervals. It was then discovered that the patient was also suffering from nasal sinusitis, but she was redischarged on April 9, 1927. Pneumothorax treatments were continued. The patient was again discharged on June 9, 1927. She was readmitted on October 12, 1927, because it was decided to supplement the pneumothorax treatment by phrenicectomy. The phrenicectomy was performed on the right side on October 22, 1927. The patient was moderately improved by this procedure. She was discharged on October 29, 1927, and readmitted on November 22, 1927. Definite pus was recovered from both maxillary antrums on November 22, 1927, when they were incised and drained. Although the patient was also suffering from involvement of her sphenoidal and ethmoidal sinuses, it was decided that there was no indication for investigating them at that time.

The improvement which followed the drainage of both maxillary sinuses was striking. This improvement has been maintained. It is obvious that the associated treatments were responsible for some of the improvement. That is beside the point. The important consideration is the fact that the operation on the nasal sinuses contributed to this improvement.

Although somewhat aside from the point of this article it is of interest that the toxic thyroid symptoms also subsided. In the light of the important work of Cole and Womack on the experimental production of the toxic thyroid state by infections of various kinds in the body, it seems probable that the disappearance of the thyroid symptoms is to be explained, in part at least, by the general improvement in the patient's sepsis.

The results are not always so gratifying, as some of the following reports indicate.

CASE 2.—Bronchiectasis: nasal sinusitis.

Treatment: Drainage of nasal sinuses, pneumothorax treatment (right), phrenicectomy (right).

R. H., female, aged twenty, was admitted to Barnes Hospital on June 28, 1928, complaining of chronic nasal discharge, hoarseness, cough and foul expectoration. Personal history: pneumonia at the age of twelve, ill for one month; tonsillectomy in 1927. The patient's right and left ethmoidal and sphenoidal sinuses were drained on June 28, 1928. The latter were found to be filled with pus. A roentgen ray film of the chest on June 30, 1928, suggested bronchiectasis. The patient was discharged, however, on July 1, 1928.

The patient was readmitted March 18, 1929. More attention was paid to her chest condition and a lipiodol injection was performed. The diagnosis of a rather advanced right-sided bronchiectasis, with early bronchiectasis on the left side, was substantiated. Pneumothorax treatment, right, was started and was continued at intervals. Less than six months had elapsed before it was found that pneumothorax was no longer possible because of adhesions. A phrenicectomy, right, was performed on November 7, 1929. It was not followed by much rise of the diaphragm. The physical findings have up to the present remained unaltered. The lipiodol picture likewise. The patient's cough and expectoration were only somewhat reduced; in reality she benefited but little from the pneumothorax treatment, nasal sinus operation and phrenicectomy. As far as the patient is concerned, however, this improvement is sufficient to have justified the operations. They were considered worth while because even slight relief is welcome.

CASE 3.—Bronchiectasis, left.

Treatment: Phrenicectomy (left), pneumothorax (left), neosalvarsan injections.

C. W., male, aged eighteen, was admitted to Barnes Hospital on February 17, 1929, suffering from a typical left-sided bronchiectasis, probably of influenzal origin apparently starting in 1918. Cough was the patient's only complaint during the years 1918 to 1927. Expectoration had been present only since 1927 and followed an attack of left-sided dry pleurisy.

On admission the patient was found to be raising between 120 and 180 c.c. of whitish mucopurulent, occasionally blood streaked, sputum. A phrenicectomy, left, had been performed elsewhere in January, 1928. There was no improvement following this procedure. A lipiodol injection on February 22, 1929, showed a well-marked left lower lobe bronchiectasis. The dilatation extended well over the stomach bubble and out to the chest wall. A second lipiodol injection on February 25, 1929, failed to reveal any gross evidence of right-sided bronchiectasis. Pneumothorax treatment, left, was started on March 5, 1929, and was continued at weekly intervals, being supplemented by the administration of neosalvarsan. The patient was found to be suffering from maxillary sinusitis and so a Caldwell-Luc operation was performed on April 3, 1929, under general anesthesia. The lining membrane was very much thickened throughout. Polyps appeared generally. A lipiodol injection performed on April 26, 1929, showed that moderate compression of the affected lung was still being maintained. On June 5, 1929, the patient's sputum was only one-fourth in amount of what it had been on admission. The striking improvement was due to the combined treatment.

CASE 4.—Bronchiectasis, bilateral: chronic nasal sinusitis, bilateral.

Treatment: Drainage of nasal sinuses.

C. B., aged twenty-one, was first admitted to Barnes Hospital, May 19, 1927,

complaining of cough, pain in the left chest and expectoration, all of which followed an attack of influenza in 1920.

The patient was found to be suffering from what was considered to be a bilateral bronchiectasis and also from chronic bilateral maxillary sinusitis. Green pus was obtained from the sinuses by irrigation on May 25, 1927. Dr. Arbuckle opened up the right antrum through a Caldwell-Lue incision and found the cavity filled with a thick pyogenic membrane and pus. The anterior two-thirds of the right middle turbinate were removed and the cells opened high up. The microscopic examination of the antral contents and of a nasal polyp showed an edematous fibrous tissue infiltrated with chronic inflammatory cells. The antrum was lined by a stratified columnar epithelium. Fragments of the antral wall also showed a chronic inflammatory process. On June 8, 1927, a Caldwell-Luc operation was performed on the left maxillary sinus. The canine fossa was found to be filled with pyogenic membrane and pus. After the membrane had been removed it was then discovered that the nasal wall was chiefly membranous. On June 25, 1927, the sphenoid and posterior ethmoid cells on both sides were opened and found to be filled with granular cells. The patient was discharged and a note made by Dr. Graham six months later, and subsequent communications to him stated, that the patient was free from cough and expectoration and regarded herself as well. Strangely enough, however, a physical examination on September 17, 1928, still gave evidence through physical signs of lung involvement, particularly at the right base where there were many high-pitched râles. She later had some return of both cough and expectoration, but both she and her parents considered that there was marked improvement in her condition.

CASE 5.—Bronchiectasis, bilateral: chronic nasal sinusitis: pansinusitis (bilateral). Treatment: Bilateral compression therapy. Pneumothorax (left), phrenicectomy (right), operation on nasal sinuses.

T. L., male, aged twenty-seven, was admitted to Barnes Hospital on February 25, 1930, complaining of cough and expectoration of twelve years' duration. These complaints apparently followed an attack of influenza in 1918. Lipiodol injection revealed bronchiectasis with atelectasis of the left lower lobe. The patient was given pneumothorax treatment, left, on March 17, 1930; the initial pressure was minus 10, minus 14, 150 c.c. of air were introduced. Final pressures were minus 6, minus 2. On March 19, 1930, it was apparent that the patient had had a spontaneous pneumothorax on the left side. Compression of the lung was more complete than could be accounted for by the previous air injection. Repeated pneumothorax treatments did not improve the patient's condition very much, so that before undertaking anything more radical it was decided that the patient's nasal sinuses, which had been examined by Dr. L. W. Dean, should be treated.

A Caldwell-Luc operation, left, was performed on March 21, 1930. The antrum was found to be lined by a very thick, dirty gray, membrane. Patient's pneumothorax treatment was continued throughout this period but on April 30, 1930, it was again noted that the pneumothorax treatment had improved the patient's condition but little. A phrenicectomy, right, was performed on May 17, 1930. The entire nerve was obtained and although the right diaphragm showed paradox movement, it rose but little following this procedure. In fact, the patient had some trouble in raising his sputum for a short period after this procedure and also some slight dyspnea which has persisted. It was, therefore, decided to withdraw the air from the left pleural chest and this was accordingly done on May 26, 1930. The patient's vital capacity at this time was 2,000 c.c. He was then given several therapeutic lipiodol injections and was discharged on June 10, 1930.

He was readmitted on July 8, 1930, and on July 9 a tonsillectomy and adenoidectomy were performed. The patient was discharged on July 10 but was readmitted

on July 21, 1930. Maxillary sinus, right, was incised and drained on July 31. The patient was discharged on July 23, 1930, and was readmitted on August 4, 1930. On August 5 the ethmoidal and sphenoidal sinuses, left, were incised and drained. The mucous membrane was found to be thickened and sclerotic. The patient was discharged on August 7 but was again admitted on August 26. An August 27 the right sphenoidal and ethmoidal sinuses were incised and drained. The patient was discharged on August 31, 1930.

In reply to a letter the patient in April, 1931, stated that he did not feel that any of the operations on his nasal sinuses and upper respiratory tract had benefited him. He had continued treatment after leaving Barnes Hospital. His nose still felt stuffed, his head full and there was still copious discharge from his sinuses. His cough and expectoration were as before. Later reports stated that the patient's general condition remained unchanged.

CASE 6.—Bronchiectasis, bilateral: pansinusitis, chronic, bilateral.

Treatment: Operation on nasal sinuses; lipiodol injections.

W. J. G., male, aged thirty-two, was admitted to Barnes Hospital on April 14, 1930, because of chronic sinusitis and bilateral bronchiectasis. Patient was operated upon on April 15, 1930, by Dr. Arbuckle. The ethmoidal and sphenoidal sinuses were incised and drained. Radical operations were performed upon both maxillary sinuses. Observed in April, 1931, the patient was found to be able to breathe more freely. His head felt clearer, but his cough and expectoration were only moderately improved. It is to be noted that the patient has been receiving therapeutic lipiodol injections into his lungs every week. The sinuses were found to be filled with pus. The patient was discharged on April 19, 1930, improved. He has maintained the improved condition up to the present time.

CASE 7.—Bronchiectasis, bilateral; nasal sinusitis.

Treatment: Operation on nasal sinuses.

H. M., male, aged fifty-seven, was admitted to Barnes Hospital on March 18, 1928, complaining of cough of ten years' duration, weakness of two months' duration and fever of thirty years' duration and soreness of left shoulder and back of seven years' duration. The onset of the present illness is uncertain but dates back to when the patient was twenty-five years of age.

Physical examination revealed a definite left lower lobe bronchiectasis, and a phrenicectomy was performed March 23, 1929, but was not followed by any improvement. Dr. Arbuckle performed a Caldwell-Luc operation on April 2, 1929, on the left side and found the antrum lined with pus and thick pyogenic membrane. On April 16, 1929, the operation was repeated on the right side and the ethmoid and sphenoid sinuses were incised and drained. The antral mucous membrane was very hyperplastic, not polypoid. There was no pus. On April 24, 1929, it was found that the patient was definitely sensitive to lettuce, chicken, pork, duck and wool. The patient died on June 23, 1929, from a left lower lobe pneumonia without showing any improvement at any time following the operations.

CASE 8.—Bronchiectasis, bilateral.

Treatment: Phrenicectomy (left), operation on nasal sinuses.

G. M., male, twenty-two years old, was admitted to Barnes Hospital, September 26, 1928, complaining of cough, expectoration, weakness, dyspnea, soreness of the right chest and poor appetite. The patient had an appendectomy elsewhere in 1921 which had been followed by what was considered to be a postoperative pneumonia but which subsequently proved to be a foreign body lung abscess. Dr. Chevalier Jackson removed from the left lung a metal band which had been on a tooth. All this took place apparently shortly after the appendectomy. Two weeks after the bronchoscopy,

the patient developed an empyema thoracis on the right side and within three months he was subjected to three operations. The exact nature of these operations could not be ascertained but two of them were rib resections. The striking complaint at the time of his admission to Barnes Hospital was the copious, foul, occasionally blood streaked, expectoration. Physical examination and a lipiodol injection revealed a definite bilateral bronchiectasis.

The patient would not submit to treatment and so was discharged on September 30, 1928, only to be readmitted on October 1, 1928. A phrenicectomy was performed on October 3, 1928. The left diaphragm did not rise but became immobile. The roentgen ray film of the patient's nasal sinuses on October 4, 1928, revealed a pansinusitis. A radical maxillary sinus operation, right and left, was performed on October 30, 1928. The left and right sinuses were found to be filled with polypoid tissue and pus. As already noted, this patient was not over responsive and was discharged on November 9, 1928. He derived no benefit from his phrenicectomy. His cough and expectoration were not improved by the nasal sinus operation according to later reports.

DISCUSSION

It is admitted from the start that no operation on the nasal sinuses will cure a true case of bronchiectasis, that is, one which has been demonstrated by a lipiodol injection. In the case of bronchorrhea without bronchiectasis one may obtain marked improvement because the cough and expectoration in such a case may be entirely due to the nasal sinus suppuration. The physical findings in the lung in true bronchiectasis associated with nasal sinusitis usually remain unchanged following operation on the nasal sinus unless the patient is receiving postural drainage treatment. The size, shape and distribution of the dilatations also remain unchanged in practically every instance.

We have little proof that nasal sinusitis may cause bronchiectasis. It may indirectly be considered as a cause since it may be the cause of a rather severe purulent bronchitis, and since putrid bronchitis may eventually terminate in bronchiectasis. On the contrary, also, there is little proof at the present time to substantiate the suggestion that bronchiectasis is a cause of nasal sinusitis. And so, let us repeat again that we do not suggest that an operation on the nasal sinuses will cure patients with bronchiectasis. It will influence the cough and expectoration in many cases. Operations on nasal sinuses are therefore indicated in many cases since any improvement is worth while in a case of advanced bronchiectasis. Correction of the nasal sinuses should always be undertaken before any serious operation is undertaken upon the lung proper, particularly when one is dealing with bilateral disease. Statistics which attempt to show the value of treating nasal sinus disease in association with bronchiectasis are misleading. They are misleading because the operation on the nasal sinuses is usually one of many treatments which have been employed and it is difficult to determine in many instances whether or not the beneficial reaction is not the result of the combined treatment. It is not unlikely that the nasal sinus suppuration develops

at the same time that bronchiectasis does in many cases. When all the sinuses are involved it is usually customary to treat one sinus at a time, subjecting the patient to as little trauma as possible. In some cases osteomyelitis of the bony wall of the sinus renders a good result impossible (Dean). Perhaps the results will be more striking if the nasal sinuses are corrected early during the course of the disease, that is, during childhood. It may be possible to determine which type of bronchiectasis is most frequently associated with nasal sinus suppuration and which type seems to respond best to the treatment of nasal sinuses. Since the use of lipiodol is relatively recent, it will be impossible to make really trustworthy observations in this direction for some time to come.

(To be continued)

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts*

Hedenius, I.: Complications pleuro-pulmonaires dans certaines affections abdominales. (Pleuropulmonary Complications in Abdominal Lesions.) Acta. Med. Scandinav. **76:** 19, 1931.

A study is made of the pleuropulmonary manifestations secondary to acute disease of the upper abdominal organs. The upper abdominal lesions most frequently giving rise to pleuropulmonary lesions are: (1) Acute, particularly purulent, infections of the biliary tract, (2) acute pancreatitis, (3) peri-gastritis with abscess, secondary to perforations of peptic ulcers or gastric carcinomata.

Pleuropulmonary complications are far more common following lesions of a biliary tract than of the other groups. Three cases are reported illustrative of these complications. The most frequent complication is a right-sided pleuritis complicating acute purulent cholecystitis or chronic empyema of the gall bladder. The pleural exudate is usually serous and usually recedes following an evacuating aspiration. They are analogous to the pleural exudates seen in the vicinity of encapsulated purulent pleural exudates notably near metapneumonic intralobar empyemata.

Contrary to the metacholecystitic complications, the pleuropulmonary lesions complicating acute pancreatitis are usually situated on the left side. They present the same tendency to recede and are occasionally chylous.

Considering the pathogenesis of these lesions, the following may be stated: A relative immobility of the diaphragm is present in the acute crises of acute cholecystitis and acute pancreatitis which results in an incomplete atelectasis of the inferior border of the lung. The dorsal decubitus is an adjuvant to this condition and facilitates infection of the atelectatic portions by way of the bronchial tree. The fact that the complications of acute cholecystitis are usually on the right whereas those of acute pancreatitis are usually on the left would seem to weaken this hypothesis. It may be that the complications of pancreatitis arise by retroperitoneal infection. An upward spreading retroperitoneal lymphangitis can involve the left pleura by contiguity anatomically more easily than the right.

The pleuropulmonary complications of esophageal lesions are rare. An ulcerating carcinoma of the esophagus because of its chronicity initially causes mediastinitis which progresses to an advanced degree before pleural complications take place. Likewise because of its chronicity it is uncommon for a gastric carcinoma to be complicated by a left-sided pleural involvement. Pleuritis is much rarer in these cases than in acute biliary or pancreatic lesions.

The diagnosis of the pleuropulmonary complications often is difficult. Cough and alteration in the respiratory cycle are the most reliable signs. Usually they arise sometime after the onset of a primary acute upper abdominal lesion. The

*The abstracts in this issue were compiled by Dr. M. M. Berck, Research Fellow in Thoracic Surgery, Washington University School of Medicine, St. Louis.

evacuating aspiration of the pleural effusions usually suffices to cure the lesions but in certain cases of purulent alteration of the fluid operative measures are necessary.

Schneider, H.: Zur Frage der mechanischen Behinderung des Herzens. Ein Fall von Perikarditis tuberkulosa. (Mechanical Embarrassment of the Heart. Case of Tuberculous Pericarditis.) Deutsche Ztschr. f. Chir. **231**: 567, 1931.

In the course of pericarditis changes take place in the pericardium that threaten life through the effect they exert upon circulation by hindering cardiac action. A surgical effort is worth while to remove inflammatory exudate or to free the heart from cord-like or plaque-like imprisoning adhesions. The case cited is that of a boy of eleven, known to have tuberculous pericarditis which became a tremendous hindrance to the right heart action causing symptoms of right heart failure, cyanosis, dyspnea, edema, ascites and liver enlargement. Repeated pericardial aspirations yielding clear fluid in which no tubercle bacilli were found occasioned considerable relief, but finally pericardial aspiraton were unsuccessful for fluid while the symptoms recurred with progressive intensity. Other tuberculous lesions were not found to be present. The ascites was suspected of being tuberculous because it did not regress with pericardial aspirations when the other phenomena of circulatory failure were ameliorated. It was thought that the activity of the lesion was minimal and that the mechanical burden of the heart was the only problem. A pericardiolysis (Rehn) was therefore performed. Very thick pericardial adhesions, and to the surprise of the operator, free fluid and caseous masses lying between the epicardium and pericardium were found. The adhesive pericardial process was especially marked around the right heart being particularly firm and fixed at the entrance of the inferior vena cava. The left ventricle was not bound down and the pericardial process other than that about the right heart was not particularly severe. The epicardium over the right heart was resected with the pericardium. During the course of the operation the right ventricle was entered but the rent was sutured satisfactorily. The right pleura was entered inadvertently also. Postoperatively an appreciable improvement in the cardiac status took place, the dyspnea was greatly relieved, the cyanosis disappeared, diuresis set in and the liver grew progressively smaller. The wound broke down and the heart lay almost free in the chest wall beating in sight. The patient died seven weeks later with empyema. The operative observations indicate that circulatory failure due to pericardial adhesions can be due to adhesions almost solely confined to the right heart especially in the region of the entrance of the inferior vena cava into the right auricle. Contrary to previous observations, widespread productive pericardial lesions hindering cardiac actions may be accompanied by acute exudative changes. To this circumstance is ascribed the unfortunate postoperative course with the breaking down of the wound and the empyema.

Sauerbruch: Operativ geheiltes Aneurysma des Herzens. (Operative Cure of Cardiac Aneurysm.) Deutsche Ges. f. Chir. Sitzg. **8**: 11.IV. 1931.

Because of a case previously observed by Sauerbruch in which many of the features were similar to those observed in this case, operation for a supposed mediastinal hematoma was performed. The case previously observed had demonstrated by x-ray what appeared to be a mediastinal cyst which produced the clinical symptoms of cardiac compression by contiguity. At operation in that case a large organized hematoma adjoining the right heart wall had been found. In examination of the second patient one was led to assume both from the x-ray findings and the preliminary operative findings that a somewhat similar condition was present. A

closer scrutiny of the history and x-ray plates of the second patient might have averted the error in diagnosis. At any event with a type of cardiac disability such as was presented operative interference seemed indicated. At operation a large tumor in the juxta-cardiac area was palpated and thought to be a mediastinal cyst. There were no signs of pulsation and an aspirating needle withdrew no blood. With further manipulations the operator suddenly found that his forefinger had entered the right ventricle out of which there gushed a terrific hemorrhage. Using the entering finger as a tampon it was kept within the ring of the perforation which effectively blocked the hemorrhage, three sutures were passed around the finger finally closing off the opening. The aneurysmal sac was then incised and emptied of its thrombus. A smooth convalescence followed although there remains the myocarditis which was the antecedent etiology for the scarring, weakening and aneurysm formation.

Jansson, G.: Beitrag zur Röntgendiagnostik beim Pericarddivertikel. (X-ray Diagnosis of Pericardial Diverticulum.) Acta. radiol. **12**: 50, 1931.

A roentgenologic study of a case of pericardial diverticulum is presented. The shape of the pericardiac shadow, thought to be a tumor or exudate, was seen to vary markedly in size and shape during the phases of respiration. During deep inspiration it became elongated, narrow and somewhat tubular, parallel to the mediastinum, whereas in forced expiration it became spheroidal, wider and appreciably shorter. The mass distinctly gave the impression of something malleable, soft, expansible and yielding, rather than that of a fixed, hard exudate or tumor. This roentgen sign has not been hitherto described in mediastinal diagnosis.

Zaaijer, H.: Beiträge zu und Auffassungen über Thoraxchirugie und Resultate. (Contributions to and a Review of Thoracic Surgery and Its Results.) Ztschr. Tuberk. **59**: 37, 1930.

I. Pulmonary Tuberculosis: In large part surgical treatment is given to those patients who show no signs of spontaneous cure or in whom pneumothorax and phrenicectomy have been found to be unsuccessful. Therefore one is concerned with a very severe disease of which the early mortality is very high and one in which furthermore the patient dreads operation. Therefore every progression in technic that permits one to operate these patients with less psychic trauma and with less operative risk is to be welcomed. Reference is made in this connection to the method recently published by Andersen, namely, that in a unilateral thoracoplasty to leave the six ribs in the middle remaining, and then resect them sometime later under local anesthesia. The shock of the multiple rib resections and of the thoracic collapse are thus separated. It is known in this connection that the first stage of thoracoplasty has much the severest postoperative reaction. A very real hindrance in the performance of multiple stage thoracoplasties is the premature ossification arising from the periosteum left behind. Meiz has worked out a method whereby by painting the remaining periosteum with formalin the ossification arising from it will be arrested or, at least retarded. By the combination of these procedures one can perform a thoracoplasty by as many stages as the condition of the patient or the lesion demands, and one can interrupt the operation at any point without fear of vitiating the end-result. Using this method the author recently operated upon three very ill patients and was astonished to see how well they withstood the procedure. One is cured, another still under treatment though improved and the third dead after an exacerbation of his lesions in the opposite lung, seven months after operation. In order not to unduly disturb the psyche by the frequent operative repetitions, the author prefers general nitrous oxide anesthesia to local infiltration anesthesia. General anesthesia is much more easily borne by the patients and

considerably reduces the dread of the repeated operations. A small admixture of chloroform deepens the anesthesia and has been found to be harmless. The caution is expressed that the mixture must be given by an experienced anesthetist. In over four thousand cases of nitrous oxide anesthesias there have been only six respiratory arrests; two deaths could not be ascribed to the anesthesia, one being in a case of esophageal carcinoma and the other in an anemic, highly febrile, patient with a thoracic fistula.

II. Bronchiectasis: The number of cases of bronchiectasis coming to surgery is on the increase. One reason may be that the differential diagnosis between tuberculosis and bronchiectasis has been refined. Nearly all the bronchiectasis cases in Holland give a history of having been under treatment in a tuberculosis sanatorium. Doubtlessly many more such are at present under such treatment. In this connection the figures of Renden are illustrative; which show that only 25 per cent of patients in sanatoria at present in Holland show tubercle bacilli in the sputum. A study has been made of the results with phrenicectomy in bronchiectasis. In two recent cases extraordinary improvement is reported; one in which the right lower half of the lobe had inflammatory bronchiectasis and in which expectoration has practically ceased with high fixation of the diaphragm, and the other a case of congenital bronchiectasis of the left lower lobe which is now fully arrested. One must consider phrenicectomy in cases of bronchiectasis as more than merely a preparatory operation.

In consideration of other operations, the author, in spite of his own inexperience with the method, considers plombage as too hazardous. Thoracoplasty with extensive rib resection with the periosteum over the site of the lesion is recommended for some cases. This method sufficed for two cases, which now after ten to sixteen years can be pronounced permanently cured. Removal of the periosteum and the intercostal musculature with the ribs is very important in these cases in spite of the danger of mediastinal flutter. The employment of the measures recommended above for thoracoplasty in tuberculosis will prove of service also with these cases.

In lobectomy it has been shown that it is very important to fix the healthy lung to the chest wall by adhesions so as to blockade the general pleural cavity from the contamination subsequent to amputation of the lobe.

Stegeman emphasized that general anesthesia in thoracoplasty is of great importance. He prefers narcylene in spite of the explosive danger because of: (1) lower mortality; (2) quicker induction and recovery; (3) Less postoperative nausea and emesis; (4) minimal effects upon the respiratory organs.

Denk has used phrenicectomy for bronchiectasis for the past nine years with good results.

Hauke, H.: Fehler und Gefahren bei der Thoracoplastik. (Mistakes and Failures of Thoracoplasty.) Brun's Beitr. **151**: 615, 1931.

The personal experience with 200 cases of thoracoplasty is the basis of the paper. Analysis is made of the chief contraindications of the operation. The author strongly insists upon the nonoperability of those with mobile mediastina and excessive emphysema on the sound side. Preference is made for patients under thirty years of age. More recently the cases, with lesions strictly limited to an upper lobe, have been initially treated with pneumolysis and plombe. Preference is given to these procedures in these cases before thoracoplasty is attempted. The author stresses individualization of each case with reference to the sites and amounts of ribs resected, particularly in the resection of the upper ribs. The employment of thoracoplasty has not met with uniform success in the treatment of abscess and bronchiectasis. The author concludes that mediastinal flutter is the chief hazard of thoracoplasty and that the fixed fibrotic mediastinum is the greatest adjuvant

for the success of the operation. A statistical summary of the 200 cases shows 40 per cent restored to earning a livelihood and 35 per cent clinically improved.

Schedtler: Statistik und Ergebnis über Thoracoplastik. (Statistical Summary of the Results With Extrapleural Thoracoplasty.) Deutsche med. Wchnschr. **57:** 491, March, 1931.

The operative indications for thoracoplasty are:

1. Well localized, sclerotic tuberculous cavities.
2. In bilateral tuberculosis when one of the lungs is not too seriously damaged and the other (the "better" side) shows no signs of progression after two months of observation.
3. Incomplete pneumothorax with small, apical, adherent cavities.
4. Pneumothorax with serous or purulent exudate.
5. Residual cavities of large pyopneumothoraces impossible to close with other means.
6. Serious hemorrhage when pneumothorax is impossible or inefficacious.
7. Sclerotic tuberculous pneumonitis with resultant cardiac distress from displacement (acquired dextrocardia).

The technic employed by the author consists in:

1. Physical and psychical "building up" of the patient.
2. Local anesthesia progressive with the operation.
3. Rapid costal resections.
4. Small rubber drain for twenty-four hours.
5. Compression dressing.
6. If the operation is done in two stages an interval of three weeks intervenes.
7. Postoperative exercises to avoid scoliosis.

Statistics: (Based upon 320 extrapleural thoracoplasties of which eighty were done in one stage and 240 in two stages.) Primary Mortalities: (1) Seventeen died in the first eight postoperative weeks (5.3 per cent); of these seventeen, nine died of tuberculous complications. However, in 239 cases operated upon for unilateral cavernous tuberculosis only eight died.

2. Statistics of Bull: 401 cases—11 per cent mortality.
3. Statistics of Alexander: 1,159 cases—12.3 per cent mortality. Late Results—(Author's Series).

Of the 200 cases operated upon between 1914 and 1929—129 still are living of whom 87 are cured—67 are dead—4 have not been traced.

It is difficult to compare the statistics of different operative clinics because of the variations in the interpretations of operative indications.

Kirschner, R.: Ein Verfahren, die unmittelbaren schädlichen Einwirkungen der Thoracoplastik auf Atmung und Kreislauf zu mindern. (A Method to Lessen the Immediate Harmful Effects of Thoracoplasty Upon Circulation and Respiration.) Deutsch. ges. f. Chir. Sitzg. v. 8-11. IV. 1931.

From the patient's viewpoint the most important immediate postoperative complication is the pain dependent upon the movements of the ends of the severed ribs. The musculature has lost a large area of its points of insertion. A very important complication may therefore ensue in paradoxical motion of the ribs with harmful effects on the mediastinum with the consequence of mediastinal flutter.

It is important and necessary to be able to predicate the amount of collapse to be given by thoracoplasty and to be able to arrest it from progressing too far. It is also important to be able to maintain the desired amount of collapse. Some cases show a much greater degree of thoracic contraction after thoracoplasty than others. To secure a uniformity of maintenance of the optimum degree of collapse—the author

uses the following procedure; the vertebral ends of the first eleven ribs are severed but only the first, second, third, fifth, sixth, eighth and ninth are resected; the fourth, seventh, and tenth are only cut through. The thorax now can collapse. That it may be fixed in the optimum position of collapse the segments of the merely severed ribs are then firmly fixed by suture to each other, being carefully approximated with bone forceps and firmly held by suture in the position as seems desirable for the degree of collapse sought for. One can therefore predicate the exact degree of collapse that will be maintained despite further contraction or expansion and keep the hemithorax fixed in the optimum position. One need not now wait for secondary contracture to complete the collapse but may attain immediately the desired result. To a certain extent one can therefore perform an individualized selective thoracoplasty.

The author does not approve of general anesthesia but instead prefers spinal anesthesia. This provides anesthesia up to the jugular notch. The resection of the first rib is performed under local infiltration percain anesthesia.

Denk: The collapse produced by Kirschner's method is not sufficiently great. He considers the uniformity of collapse more important than the absolute degree of it.

Hauke: Uses the paraffin plombe extrapleural pneumolysis technic for the collapse of the upper lobes. Up to the present he has had about thirty-two cases of this. He does not think Kirschner's method will attain its objective.

Iselin, M.: La Phrenicotomie Bilaterale et les Problemes Physiologigues qu'elle pose (Bilateral Phrenicectomy and Its Physiological Problems.) La Medicine, **12**: 389, May, 1931.

The frequent practice of phrenicectomy in pulmonary tuberculosis has modified considerably the concepts of the physiology of the diaphragm and its active importance in respiration. It had been shown irrefutably that the phrenic is the sole motor nerve to the diaphragm and the lower intercostal nerves serve it merely as sensory pathways. The diaphragm as a muscle of respiration has no active rôle, serving merely to maintain the vertical diameter of the thoracic cage, and indeed when it is paralyzed it is aspirated upward by the negative intrathoracic pressure, being at the same time pushed upward by the intraabdominal pressure.

The diaphragm may be considered as a membrane separating the thoracic and abdominal cavities. When paralyzed it rises conforming to the status of the lung. If the lung is but slightly affected the rise is immediate, if the lung is densely infiltrated, the ascension may be minimal in spite of the intraabdominal pressure. When elevation ensues several months after phrenicectomy, it is pulled upward consequent to the cicatricial retraction of the lung.

In spite of the apparent inocuousness of unilateral phrenicectomy—bilateral diaphragmatic paralysis has been regarded as dangerous. Jehn, during the war, performed bilateral phrenicectomy for the diaphragmatic spasms of asphyxial tetanus. The paralysis of the diaphragm permitted the performance of prolonged artificial respiration and enabled him to save a few of these patients. Krohn performed the operation for the same indications. But a long time elapsed before it was applied to tuberculosis. Brunner had recourse to the operation in a peculiar instance. After a thoracoplasty preceded by phrenicotomy the patient developed a hiccup which became serious. The phrenic of the opposite side was cocainized with immediate relief. The patient recovered with a slight dyspnea. But the cases have been few because bilateral tuberculosis has not been considered surgical and general opinion has deemed such a procedure dangerous and its good effects, if any, transient. There is, however, the observation on a child operated upon by Sauerbruch in whom three and a half years later it was noted that both diaphragmatic leaves were highly

elevated and immobile, the amplitude of a thoracic cage was nearly normal, the bilateral pulmonary tuberculosis was healed, the child appeared normal and normally followed the gymnastic activities of his schoolboy comrades. Curti has performed six bilateral phrenicectomies in children with bilateral acute severe pulmonary tuberculosis. The results of these were satisfactory in that there were two great improvements, three moderate improvements and one death. This author restricts the operation to particularly severe cases. The nerve resections were performed simultaneously in five of the cases. In one case fifteen days elapsed between the two operations.

The published cases have, of course, been too few to allow an evaluation of the method. Up to the present most of them have been performed in children with acute tuberculosis. Perhaps children can compensate for the loss of the diaphragm more easily than adults because of the suppleness of the infantile thoracic cage. Cardis and Malinsky have reported two good results in adult chronic lesions.

Naegeli, T., Schulte-Tigges, H.: Die radikale Phrenicusoperation und ihre Bedeutung bei der chirurgischen Behandlung der Lungentuberkulose. (The Value of Phrenicexeresis in the Surgical Treatment of Pulmonary Tuberculosis.) Ztschr. Tuberk. **59**: 37, 1930.

This report of phrenicexeresis as an independent procedure in the treatment of pulmonary tuberculosis is related to that of the same authors published in 1927 wherein it was reported that 48 per cent of the cases with cavity formation were rendered free of bacilli. In this report some of the cases previously operated upon are again reviewed with the more recent cases operated upon in the past four years. Fifty-five cases are the basis of the report. Of these 30.8 per cent are bacilli-free and capable of working; 43.7 per cent had good results, the cavities considerably shrunken but bacilli still present in the sputum. A complete cure in this last group would probably be effected with further collapse measures. Twenty-five and four-tenths per cent had become appreciably worse.

The aversion of general surgeons to phrenicexeresis as the sole measure of collapsotherapy in pulmonary tuberculosis is comprenhensible in view of the fact that the cases usually seen by them have severe, widespread lesions. The sanatorium physicians, on the other hand, can furnish more examples of good results following phrenicexeresis as an independent and sole procedure because of the varied material and milder type of case often seen by them.

Felix, W.: Beitrag zur intrapleuralen Pneumolyse. (Report on Intrapleural Pneumolysis.) Zentralbl. f. Chir. 1931 **26**: p. 1660.

The author reports four cases of pulmonary tuberculosis in which pneumothorax was unsuccessful because of pleural adhesions and in which intrapleural pneumolysis was performed by the open method. With all the patients at least four adhesions were present, some as thick as one's thumb. Operations as performed consisted of the resection of the third and fourth ribs posteriorly (the adhesions were all apical) and the severance of the adhesions under good vision with diathermy. Thick adhesions were clamped and ligated before severing. Thinner adhesions were severed after preliminary pressure with long clamps. The advantages claimed for the open method over the closed (Jacobeus) technic are: (1) Full vision and good orientation in the operative field. (2) Complete certain hemostasis. (3) Shortened operating time.

The disadvantage of the open method as compared with the Jacobeus technic is that it is a much more formidable operation. Two of the four patients became sputum and fever free, the two others in whom pneumothorax had been present for

a longer time had a diminution in the sputum and in the bacillary count. Twice a transient subcutaneous emphysema became evident over the shoulder girdle and face. All four developed a sterile pleural exudate.

Kremer: Surgical severance of pleural adhesions was first proposed twenty years ago but has not been taken up with enthusiasm. Only twenty cases had been reported up until 1925. At present over two thousand cases, treated by the Jacobeus technic, have been reported. Only numerous operations can decide whether the open or closed technic is the better, until at least five hundred cases have been treated by the open method. It is true that all the adhesions usually cannot be severed in one session with the closed technic. There have been occasions when twenty-five adhesions have been severed and still more remained. As far as hemostasis is concerned, in the last three hundred cases treated by the speaker with the closed technic, no difficulty has been encountered on that score. It is true that orientation within the thorax with closed technic is difficult but the speaker's modified system of lenses facilitates matters.

Vidakovits, C.: Zur Technik der Extrapleuralen Pneumolyse. (Technique for Extrapleural Pneumolysis.) Zentralbl. f. Chir. **58:** 1496, June, 1931.

Although in complete agreement with the indications given by Sauerbruch for extrapleural pneumolysis and plombage, one cannot always unconditionally adhere to them. Increasing experience is teaching us that the indications for the operation should be widened. The very great fear of overflow aspiration infection, in cases in which a good compression of a large cavity can be secured and in which there is not too copious sputum, is unfounded. Many cases have now been observed which formerly would have been treated with thoracoplasty which have gone on to improvement and cure with extrapleural plombage. One is impelled from the consideration of these cases to place plombage well into the foreground in the treatment of unilateral upper lobe tuberculosis.

The author proposes a new skin incision for pneumolysis, impelled to do so for cosmetic reasons. Patients have often refused thoracoplasty because of the large scar and the resulting deformity. The laity has become acquainted with the cosmetic results of thoracoplasty and recently patients have come asking for plombage and unwilling, under any circumstances, to undergo thoracoplasty. Moreover many will not avail themselves of plombage because of the resultant scar. The author has considered the possible incisions for apical pneumolysis: (1) The supraclavicular, which is not practicable; (2) the infraclavicular of Tuffier. The advantages of this approach are the ease and simplicity of access to the upper lobe and that it can be performed without rib resection. Its only disadvantage is the large cosmetic deformity. The author has had no experience with this incision rejecting it because of the deformity. (3) The axillary incision. The disadvantages here are: (a) The operation is performed in a site where trustworthy skin disinfection is difficult; (b) The approach to the apex is not any better than the subclavicular route, in fact is often worse and does not give as easy access to the costovertebral gutter; (c) No muscle is present for closing over the defect produced.

(4) The posterior approach of Baer is approved because of: (a) Ease of access to all parts of the upper lobe; (b) aseptic operating field; (c) abundance of musculature in layers to close in over the defect. This procedure is advocated with two changes in the incision. The incision is made lower down and is curved. The most superficial muscle layer is cut through transversely and the deeper layers are divided in the corresponding layers of their fibers. The skin incision begins at the border of the scapula approximately opposite the origin of the third rib and runs in a convex curve downward. The skin flap is dissected up widely particularly toward the midline where it is very loosely attached. Only the superficial layer of

muscles is cut through transversely. The rest of the operation is as has been described on other occasions. The proposed incision leaves a level surface on the upper part of the back with the scar well down out of sight which is welcomed by the patient and encourages others to have the operation performed, the rib defect is covered in by thick layers of muscles, the skin and muscle incisions do not overlap and the deeper muscles are undisturbed.

Lotheissen, G.: Diagnose und Behandlung der Divertikel der Speiseröhre. (Diagnosis and Treatment of Esophageal Diverticulae.) Klin. Wchnschr. p. 73, 1931.

The article is based upon an analysis of 60 operative cases of esophageal diverticulum. The early symptoms of esophageal diverticulum are not characteristic. Sometime later, however, the following characteristic symptoms appear; frequent expectoration of tenacious mucus, scratching sensations in the lower throat, frequent annoying cough, a sense of fullness and pressure with solid food, then later vomiting without nausea. A well taken history often points the way to a correct diagnosis. One may pass a stomach tube and attempt to aspirate the pouch. For this purpose a curved blunt-tipped tube is used. By all means, however, the best diagnostic measure is the fluoroscopic and x-ray demonstration of the diverticulum with contrast media.

Only surgical treatment can cure the condition. If operative treatment is refused, dilatation occasionally ameliorates. Certain cases of deeply situated thoracic diverticula are often associated with cardiospasm. Such cases respond well to dilatation treatment. The toal extirpation of the sac is the only procedure for permanent cure. Preliminary gastrostomy is necessary only in the very badly deteriorated cases. Of a series of 191 cases refusing operation or for other reasons being nonsurgically treated, 23 died from starvation or pulmonary complications consequent to aspiration from the diverticulum. The mortality of the 60 cases treated by a one stage operative technic was 1.6 per cent.

Operative interference should not be undertaken in bad risk patients unless the symptoms are unbearably severe. Traction diverticula are very hazardous to operate upon because of the dangers of damage and contamination of the neighboring thoracic structures.

Wilder, T. S., and Stokes, J.: Esophageal Fistula in the Experimental Animal. Arch. Surg. 23: 1, July, 1931.

The authors review the literature concerning the effects of esophageal obstruction and fistulization. Recent workers have produced esophageal obstruction in dogs with rapidly fatal results, their chemical studies showing a decrease in plasma chlorides and an increase in nonprotein nitrogen. Wangensteen and Chum reported a rapid lethal outcome following the operation but felt that the administration of saline prolonged life. More recently the death of animals after complete esophageal obstruction in an average of 15 hours has been reported; similarly animals of complete esophageal fistulae died in an average of seventeen hours. The administration of saline and base seemed valueless. It was intimated that the loss of saliva may play an important rôle in the lethal outcome.

It is possible that technical difficulties in producing low esophageal obstruction and fistula may play a part in the high mortality previously reported. The mere lack of saliva is not particularly harmful as is shown by the citation of clinical cases of the complete absence of salivary glands and the results of experimental salivary gland extirpation in dogs. A case of complete esophageal

obstruction in a child is cited. Extreme malnutrition and underdevelopment were present which rapidly yielded to high caloric gastrostomy feedings even without salivary admixture. Experimentally produced esophageal fistula, performed in three stages, in the young pig, causing complete loss of the saliva, were found to be without material influence on normal growth. No instances of rapid death were encountered. Careful dietetic management by feedings into the distal portions of the divided esophagus was maintained. The authors state that the difference in their results from the report work on dogs may lie in the use of the three stage operation or in the use of a different animal. In these animals (one-month-old pigs) experimentally produced esophageal fistula, causing complete loss of saliva, did not materially interfere with normal growth provided that proper dietary management through the distal portions of the esophagus was maintained.

Kriszty, E.: Ein operierter Fall von Herzverletzung. (An Operated Case of Cardiac Injury.) Zentralbl. f. Chir. p. 76, 1931.

Cardiac injuries come about both through pointed and blunt instruments. With small wounds the cardiac muscle can adherently contract to a sufficient degree to stem the hemorrhage. Needles, bony splinters and projectiles can remain over a long period of time in the cardiac walls without occasioning any disturbance. If the bundle of His is injured, death may be immediate. Wounds involving the coronary artery have a very unfavorable prognosis, as have simultaneous wounds of both anterior and posterior walls. The symptoms of cardiac wounds, as well known, are: cyanosis, a small-volumed, irregular and uneven pulse, at times barely perceptible; rapid, superficial respirations; coolness of the face and extremities with clammy sweat over the hands, feet and face. In severe cases the appearance is that of a shocking anemia. The most important factor is hemopericardium with cardiac tamponade. Operation should be performed for cardiac injury when the wound has been infected through penetration of the wounding instrument through an infected area; when there is the danger of embolization of projectiles; or when the situation of foreign bodies is such that complications are likely to ensue later. Hemorrhage is, of course, an absolute indication for operation. Needles can be extracted from the heart muscle wall with safety. But knives and other such large wounding instruments, should not be extracted until the patient is being operated upon, and arrest of a hemorrhage from the cardiac wound can be accomplished instantaneously. Postoperative adhesions between the heart and pericardium occasionally occur of such degree as to necessitate cardiolysis.

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The Fifteenth Annual Meeting of the American Association for Thoracic Surgery

THE Fifteenth Annual Meeting of the American Association for Thoracic Surgery will be held in Ann Arbor, Michigan, on Monday, Tuesday and Wednesday, April 18, 19 and 20, 1932.

Hotel headquarters will be the "Michigan Union" of the University of Michigan. For reservations please write Dr. John Alexander, University Hospital, Ann Arbor, Michigan.

The following is the first draft of the preliminary program. It is subject to considerable change in its details. It is published now to furnish a general conception of the final program. Several papers will be added to this preliminary program, and in two instances the authors reserve permission to change the subject of their address.

Preliminary Program

"President's Address." Dr. Frederick T. Lord, Boston.

"The Innervation of the Heart. Studies on the Intrinsic and Extrinsic Nerve Supply." Dr. Peter Heinbecker, St. Louis.

"The Surgical Treatment of Carcinoma of the Lung." Dr. Edward D. Churchill, Boston.

"The Nature and Prevention of Post-Operative Pulmonary Complications in Chest Surgery." Dr. Pol N. Coryllos, New York City.

"Thoracoplasty in Tuberculosis of the Lung." Dr. Adrian V. Lambert, New York City.

"Special Consideration with regard to Closure of Large Upper Lobe Tuberculous Cavities." Dr. John Alexander, Ann Arbor.

"Surgery of the Lung." Dr. Willy Meyer, New York City.

"Tuberculous Empyema." Dr. Carl A. Hedblom, Chicago.

"The Present Status of 100 Patients Treated with Artificial Pneumothorax after One to Eighteen Years' Reexpansion of the Lung." Dr. Edward N. Packard, Saranac Lake.

"Surgical Immobilization Therapy in Tuberculosis." Drs. Evarts A. Graham, J. J. Singer, Duff Allen and Harry C. Ballon, St. Louis.

"The Treatment of Secondarily Infected Tuberculous Pyopneumothorax Cavities." Dr. Frank B. Berry, New York City.

"The Treatment of Diaphragmatic Hernia." Dr. Ralph B. Bettman, Chicago.

"The Suspended Cavity in Artificial Pneumothorax." Dr. E. J. O'Brien, Detroit.

"The Medical Treatment of Abscess of the Lung." Dr. Conrad Georg, Jr., Ann Arbor.

"The Results with Phrenicectomy in Pulmonary Tuberculosis." Drs. Lawrason Brown, J. H. Hayes and J. T. Eagan, Saranac Lake.

"Removal of a Calcified Pericardium." Dr. Howard Lilienthal, New York City.

"The Value of Endoscopy in Thoracic Surgery." Dr. William A. Hudson, Detroit.

"Bilateral Partial Thoracoplasty for Bilateral Pulmonary Tuberculosis." Dr. Duff S. Allen, St. Louis.

The final program will be published in the April issue of the JOURNAL OF THORACIC SURGERY.

Space will be available for the demonstration by members of new instruments and apparatus for thoracic surgery.